THE PLANT DISEASE REPORTER

Issued By

CROPS RESEARCH DIVISION

AGRICULTURAL RESEARCH SERVICE

UNITED STATES DEPARTMENT OF AGRICULTURE

Volume 44 Number 5

May 15, 1960

The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Crops Research Division serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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ACCEPTANCE OF MANUSCRIPTS

The increase in the volume of pertinent material offered for publication in the Plant Disease Reporter has made it necessary to limit the subject matter and the length of articles accepted. The subject matter should emphasize new things in plant pathology, such as new records of disease occurrence, serious outbreaks and epidemics, conditions affecting development of plant diseases, techniques of investigation including instrumentation, new discoveries in control including new materials and their evaluation. Manuscripts will be limited to 12-double-spaced typed pages, including tables, graphs, and photographs. Because of reproduction costs photographs should be kept to a minimum. Insofar as possible, material should be presented as graphs rather than tables. Papers cannot be accepted for publication that report routine control experiments, reviews, bibliographies without annotation, results of routine surveys, mere summaries or lists of plant diseases. By following this procedure we hope to continue publishing all articles promptly.

Paul R. Miller

Manuscripts for and correspondence about this publication should be sent to:

PLANT DISEASE REPORTER
Mycology and Plant Disease Reporting Section
Crops Protection Research Branch
Plant Industry Station
Beltsville, Maryland

THE PLANT DISEASE REPORTER

Crops Research Division
Volume 44

Plant Industry Station, Beltsville, Maryland
May 15, 1960
Number 5

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BLACK SHANK OF TOBACCO IN PENNSYLVANIA AND SOME OBSERVATIONS ON THE PATHOGEN

Gustave Silber and H. E. Heggestad[1]

Abstract

Black shank was observed in 1957 and 1958 as a disease of tobacco in Lancaster County, Pennsylvania. The pathogenicity of a culture isolated from Pennsylvania tobacco was as great as or greater than that of two cultures from different geographical parts in the United States on varieties Wilson (a Maryland type), Burley 21, Swarr-Hibshman (a Pennsylvania Broadleaf type), and Dixie Bright 101 (a flue-cured type).

BACKGROUND

Black shank, caused by Phytophthora parasitica Dast. var. nicotianae (Breda de Haan) Tucker, is one of the most destructive diseases of tobacco in southern United States. Since 1915, when this disease was first observed in the United States in the southern portion of Decatur County, Georgia, the pathogen has been distributed and established in widespread areas (10, 11). By 1922 the pathogen had infested areas of the northern and western portions of Gadsden County, Florida. Two years later black shank was noted in Alabama. Black shank was reported in Forsyth County, North Carolina in 1930 and within 5 years was present in many other counties in the State (1, 2). Tennessee and Kentucky reported black shank for the first time in 1935 (9, 12). The pathogen was believed to have been introduced into Virginia 2 years later on transplants from North Carolina, but it was not until 1939 that the disease was positively recognized in Mecklenburg County, Virginia (4, 15). By 1944 additional reports of the disease came from Halifax, Pittsylvania, Charlotte, and Franklin counties (5, 8, 13, 14, 16). In 1949 black shank was reported as a disease of Maryland tobacco (7).

Before 1951 black shank of tobacco had never been observed in the United States farther north than Maryland, but in that year the disease was noticed in Lancaster County, Pennsylvania (3). The infestation was light on one farm. In 1957 light infestations were observed on three farms and a number of growers reported that plants with a similar disease could be found on about 12 other farms. In each case the tobacco grower had secured transplants from a common local source. There was good circumstantial evidence that the pathogen had been disseminated by movement of transplants from a plant bed on one farm, although no symptoms of the disease had been visible before transplanting. The same site had been used as a plant bed for 58 years.

In contrast to the approximate 15 widely scattered infestations noted in 1957, black shank was found in only one field in 1958. The plant bed site had been treated with a fungicide, and strict sanitary measures had been followed to prevent infestation of the new site. The single infestation noted in 1958 probably had as its source of primary inoculum harvested plants in which the pathogen had overwintered. The pathogen had evidently been disseminated from tobacco stalks and leaf trash discarded near the tobacco barn after the stripping operation during the winter, as the disease was observed on tobacco plants in the path of water draining from the barnyard. The 1957 tobacco crop on this farm had been grown from transplants produced on the farm in the same plant bed that served as the source of inoculum for the other infestations reported in 1957.

MATERIALS AND METHODS

The pathogenicity of a typical isolate from diseased plants was compared with that of an isolate from diseased flue-cured tobacco in North Carolina and another pathogenic isolate used at Beltsville, Maryland. All were cultured on equal volumes of sterile corn kernels at 30° C for approximately 3 weeks; each culture was blended with 1 liter of water. The mycelial suspensions of the three isolates were used to inoculate 60 plants of the varieties Wilson (a Maryland type), Burley 21, Dixie Bright 101 (a flue-cured type), and Swarr-Hibshman (a Pennsylvania Broadleaf type) arranged in a split-plot design with three replicates. The number of plants surviving 3 weeks after inoculation was recorded.

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RESULTS

Table 1 presents the data from the inoculation trial. It is evident that the Phytophthora isolate from diseased Pennsylvania tobacco was virulent. Against the three black shank-susceptible varieties (Wilson, Burley 21, and Swarr-Hibshman) it was as virulent as the Beltsville isolate and was significantly more virulent against Swarr-Hibshman, a variety commonly grown in Pennsylvania, than was the Beltsville or the North Carolina isolate.

Table 1. Pathogenicity of three isolates of Phytophthora parasitica var. nicotianae from various geographical regions to four tobacco varieties.

Isolate	Variety	Survivors[a]	Stat. Sig.[b]
Pa.-1	Burley 21	0	a
Pa.-1	Wilson	0.3	a
Pa.-1	Swarr-Hibshman	0.3	a
Beltsville 9-56	Burley 21	3.3	ab
Beltsville 9-56	Wilson	4.0	abc
Beltsville 9-56	Swarr-Hibshman	5.3	bcd
N.C.	Wilson	5.7	bcde
N.C.	Swarr-Hibshman	8.7	def
N.C.	Burley 21	13.0	fg
Pa.-1	Dixie Bright 101	16.0	gh
Beltsville 9-56	Dixie Bright 101	18.0	h
N.C.	Dixie Bright 101	19.3	h

[a] Mean of 20 plants of each variety replicated three times.
[b] Statistical significance. Means followed by common letters are not significantly different from one another on basis of the Duncan multiple range test.

With the particular combination of varieties, inocula, and environmental conditions during the experimental period the reaction of the varieties tended to overlap. The isolate from North Carolina was less pathogenic than the other isolates. The susceptible Burley 21 inoculated with the North Carolina isolate survived to the same degree as did the resistant Dixie Bright 101 inoculated with the more pathogenic Pennsylvania or Beltsville isolate. Burley 21, however, was highly susceptible to the Pennsylvania and Beltsville isolates. Among the three susceptible varieties inoculated with the Beltsville isolate there was no appreciable difference in level of susceptibility. Though significant difference in virulence among the three isolates could not be shown with the moderately resistant Dixie Bright 101 as the suscept, the trend favored the Pennsylvania isolate as being most virulent.

DISCUSSION

The black shank outbreak that occurred in the Pennsylvania tobacco-growing area in 1957 and 1958 does not presently represent a serious problem. There was only one observation of the disease in 1958 compared with about 15 on widely scattered farms in 1957; in 1959 no observations of the disease were reported. Under the proper environmental conditions black shank could potentially become an urgent problem inasmuch as tobacco varieties currently grown in Pennsylvania are relatively susceptible.

The abrupt appearance of black shank twice in Pennsylvania with subsequent nonrecurrence of the disease is not unique. Though black shank was reported in Georgia in Mitchell County in 1928, it was not again observed in that part of the State until 1956 (6). On other occasions black shank has been known to occur in Georgia in one year but not recur for many years. The nonrecurrence of black shank in Pennsylvania can probably be attributed to poor environment, coupled with progressive crop rotation. In general, the summer temperatures are too low for rapid development of the disease.

The pattern of black shank appearance on different farms, all of which had a common source of transplants, emphasizes the importance of utilizing disease-free transplants in starting the crop. In many instances this is best accomplished by maintaining one's own plant bed site rather than by purchasing transplants grown in a different locality.

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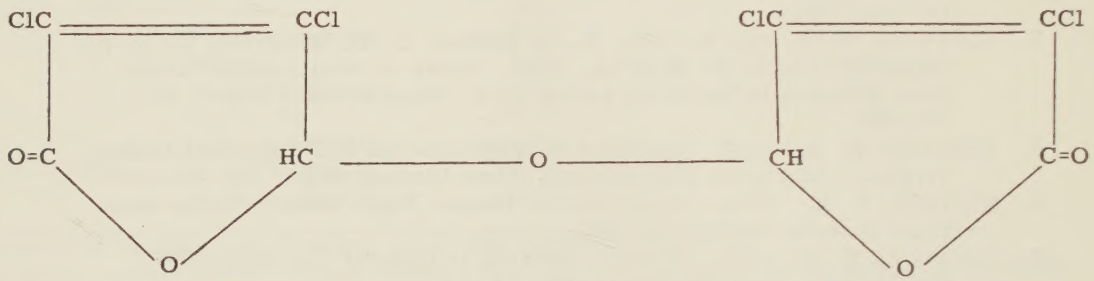
BIS-(3,4-DICHLORO-2 (5)-FURANONYL) ETHER, A PROMISING NEW NONMERCURIAL FUNGICIDE WHICH EXHIBITS BOTH PROTECTANT AND ERADICANT PROPERTIES[1]

Avery E. Rich[2]

Organic nonmercurial fungicides in which bis-(3,4-dichloro-2 (5)-furanonyl) ether is the fungicidally active component have been developed by the General Chemical Division of Allied Chemical Corporation, in cooperation with the New Hampshire Agricultural Experiment Station, under the code number GC-2466. This compound, referred to as mucochloric anhydride, has shown promising fungicidal activity in the control of certain fruit and vegetable diseases in laboratory, greenhouse, and field trials.

PHYSICAL AND CHEMICAL PROPERTIES

GC-2466 (1) is mainly the alpha isomer (m.p. 141-3° C), but a small percentage of the beta isomer (m.p. 180° C) is present. It is insoluble in hexane, petroleum ether and water, but soluble in acetone, ether and aromatic hydrocarbons. Its structural formula is:



FUNGITOXICITY

Slide germination tests, first conducted in 1953, indicate that GC-2466 has an E.D. 95 value of less than 1 ppm against Sclerotinia (Monilinia) fructicola and an E. D. 50 value of less than 1 ppm against Stemphylium sarcinaeforme.

Initial greenhouse tests (2) also showed that the chemical gave 99 to 100% control of apple scab (Venturia inaequalis) and tomato late blight (Phytophthora infestans) when applied as a protectant at 1000 ppm. The compound was then reformulated as a wettable powder, and it was retested in the greenhouse for the control of apple scab. The results are reported in Tables 1, 2, and 3.

Table 1. Comparison between captan and GC-2466, a new organic fungicide, for preinfection control of apple scab in preliminary greenhouse tests.

Chemical	Rate/100 gallons	"Rain" (in inches)	Scab infection (3plants) Plants	Leaves	% leaf area	Spray injury
		Test No. 1				
GC-2466	4 lb 25W	0	0	0	0	None
		1/2	0	0	0	do.
Captan	2 lb 50W	0	0	0	0	do.
		1/2	0	0	0	do.
Control		0	3	9	50	do.
		Test No. 2				
GC-2466	2 lb 50W	0	2	2	2	do.
		1	3	7	18	do.
Captan	2 lb 50W	0	0	0	0	do.
		1	3	8	21	do.
Control		0	3	9	51	do.

[1]Published with the approval of the Director of the New Hampshire Agricultural Experiment Station as Scientific Contribution No. 252.

[2]Plant Pathologist, New Hampshire Agricultural Experiment Station. The writer acknowledges with appreciation the helpful assistance and advice of Mr. M. M. Darley.

Table 2. The effect of reduced rate of GC-2466, captan and thiram on control of apple scab in greenhouse tests.

Chemical	Rate/100 gallons	"Rain" (in inches)	Scab infection (3 plants)		
			Plants	Leaves	% leaf area
		Protectant Test No. 1			
GC-2466	1 lb 50W	0	1	1	1
		1/2	2	2	2
	1/2 lb 50W	0	1	1	1
		1/2	3	6	6
Captan	1 lb 50W	0	0	0	0
		1/2	3	6	10
Control		0	3	12	67
		Protectant Test No. 2			
GC-2466	1 lb 50W	0	0	0	0
		1/2	1	1	3
Captan	1 lb 50W	0	1	2	2
		1/2	1	2	4
Thiram	1 lb 65W	0	0	0	0
		1/2	2	3	8
Control		0	3	9	61

Table 3. Comparison between captan and GC-2466, a new organic fungicide, for postinfection[a] control of apple scab in preliminary greenhouse tests.

Chemical	Rate/100 gallons	Scab infection (3 plants)		
		Plants	Leaves	% leaf area
	Test No. 1			
GC-2466	2 lb 50W	3	5	7
Captan	2 lb 50W	3	7	8
Control		3	9	63
	Test No. 2			
GC-2466	2 lb 50W	2	4	4
Captan	2 lb 50W	3	6	9
Control		3	9	39

[a]Fungicides applied 18 hours after inoculation with conidia of Venturia inaequalis.

The results of a greenhouse test for control of tomato late blight are shown in Table 4.

Table 4. Comparison between captan and GC-2466 for control of tomato late blight in a greenhouse test.

Chemical	Rate/100 gallons	"Rain"	% defoliation	Spray injury
GC-2466	4 lb 25W	0	0	Slight
		1/2 inch	0	Very slight
Captan	2 lb 30W	0	0	None
		1/2 inch	0	do.
Control		0	60	do.

GC-2466 was phytotoxic to peas and potato seed pieces when tested on these crops as a seed-treatment fungicide in the greenhouse.

GC-2466 was field-tested in 1957 and again in 1958 for control of apple scab and early blight of potato and tomato. The results of the apple scab tests are shown in Table 5.

In 1959 GC-2466 was field-tested at reduced rates on three timing schedules for control of apple scab. The rates, schedules, and results are reported in Table 6.

The 1-pound rate of GC-2466 (50W) adequately controlled apple scab when applied every 7 days or every 9 days in 1959 without causing excessive fruit russet.

The results of field tests for control of early blight of potato and tomato are shown in Table 7.

Table 5. Comparison between GC-2466 and other fungicides for control of apple scab in 1957 and 1958.

Treatment[a]	Year	% leaf scab			% fruit scab		
		Protect.	Weekly postinf.		Protect	Weekly postinf.	
GC-2466	1957	1			2		
Captan	do.	1			1		
Control	do.	65			98		
GC-2466	1958	0	0	0	1	0	1
Captan	do.	0	0	3		3	
Phix	do.			0			0.2
Control	do.	50			98		

[a]GC-2466 and captan were applied at the rate of 2 pounds of 50W per 100 gallons of spray during the primary infection period. Rates were reduced to 1 pound starting with second cover. Phix (22% phenyl mercuric acetate) was applied at the rate of 3 ounces per 100 gallons through first cover. One pound of captan 50W was used the remainder of the season.

Table 6. Comparison between GC-2466 at reduced levels and captan on three timing schedules for control of apple scab in 1959.

Treatment	Rate	% leaf scab			% fruit scab			
		Protect.	7-day	9-day	Protect.	7-day	9-day	Russet
GC-2466	1/2 lb 50W	5			5			Slight
GC-2466	1 lb 50W	3	2	1/2	5	2	1	do.
Captan	1 lb 50W	4			1			do.
Captan+Hg[a]	1 lb + 1 pt			T[b]			2	do.
Control		33			53			do.

[a]Puratized Agricultural, Spray.
[b]T indicates trace (less than 0.5%).

Table 7. Comparison between GC-2466 and other fungicides for control of early blight of potato and tomato in 1957 and 1958.

Treatment	Rate	% defoliation			
		Potatoes		Tomatoes	
		1957	1958	1957	1958
GC-2466	1 lb 50W		16		20
	2 lb 50W	15	16	17	25
Maneb	1 lb 65W	12	10	10	9
	2 lb 65W	10	9	9	10
COCS	3 lb	30	25	37	29
Control		66	71	40	76

The 2-pound rate of GC-2466 was slightly phytotoxic on tomatoes when applied at weekly intervals. The 1-pound rate appeared to be as effective as 2 pounds for control of early blight on both crops and it was less phytotoxic to tomatoes.

DISCUSSION AND CONCLUSIONS

GC-2466 is a potent fungicide, and it shows considerable promise for control of apple scab, either in a protectant or a postinfection schedule. More work should be done with it to ascertain the optimum dosage rate required for effective control and minimum phytotoxicity. It should also be tested further for disease control on other crops under a variety of conditions. Preliminary tests suggest that it has a slight systemic action.

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NEW HAMPSHIRE AGRICULTURAL EXPERIMENT STATION, DURHAM

DISINFESTATION OF SOME PLANTING SITES IMPROVES GROWTH OF NAVEL ORANGE TREES ON TROYER CITRANGE ROOTSTOCK

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Can Troyer citrange rootstock be used successfully in old citrus soils or in soils contaminated by microorganisms that attack citrus roots? Where this rootstock is to be used, does it pay to pretreat the planting sites to disinfest them of root-destroying fungi? Since these questions are being frequently asked, an experiment was designed to answer them for the orchard conditions described here.

Three successive plantings of navel oranges on sweet orange rootstocks in one section of an orchard[3] in Pauma Valley in 1955, 1956, and 1957 had failed (Fig. 1). The tree sites were treated with Vapam (sodium methyl dithiocarbamate)[4] prior to the 1957 plantings, but since no precautions had been taken to exclude runoff water and splashings from sprinklers they were soon recontaminated by root rot microorganisms. The soil of this area is rather heavy, being of granitic origin and containing considerable clay. Soil samples taken from this plot yielded many isolates of Phytophthora spp., the brown rot and gummosis fungi, which indicated that root rot caused by these fungi was the probable cause of failure of the trees. From 22 of the 24 tree sites chosen for the experiment, P. parasitica was isolated on February 27, 1958. One site yielded P. citrophthora. From another, neither species was recovered and the check tree planted later in that area made a good growth, which indicated the absence or low incidence of the fungi on that date. However, P. parasitica was isolated from this site on March 19, 1958.

The 24 trees sites were organized in eight groups of three sites each in order to secure the best statistical layout. One tree site in each of the eight groups was to remain untreated, one to be treated with Vapam, and one with Mylone (3,5-dimethyltetrahydro-1,3,5, 2H thiadiazine-2-thione)[4] (Fig. 2). Circular basins 8 feet in diameter and about 50 square feet in area were made around each site by ridging soil taken from within and at the exterior edge of the basin area. Removing and using soil from the outside edge of the basin made an exterior drainage trench. Through the downhill side of this trench a break was made so that surface water (from rains and sprinklers) would be conducted around and away from the treated sites and delay their recontamination. In treating with 4S Vapam, a pint of the chemical was placed in a container such as a pail or large can at the center of the site and water added to the container and allowed to overflow into the basin to a depth of 4 inches of the Vapam-water mixture. With the 85% W Mylone, 9 ounces of the powdered material was spread over the soil surface of the basin and the 4 inches of water run into a container as with Vapam.

Treated sites were dusted with 12.7% Bordeaux mixture on March 7 and on March 26, 1958. This was done to help delay recontamination of the treated basins by Phytophthora spp. While the water used on the plot was from wells and presumably free of the fungi, the basins could be recontaminated by soil bearing oospores blowing or splashing into them from the outside contaminated areas. After removal of the Bordeaux mixture on the surface, soil samples were taken from all of the treated basins on March 19 and June 12, 1958. None yielded Phytophthora spp.

After a period of 133 days to allow the chemicals to disinfest the soil and to degrade to forms noninjurious to roots, the trees[5] were planted on July 10, 1958. (A month between treatment and planting is the minimum time suggested). Around the trees small basins about 3 feet in diameter were made from treated soil within the large basins. The trees were irrigated by running water into these inner basins with a hose.

To evaluate the effect of the treatments on growth of the trees, measurements of the circumference of the trunks 6 inches above the bud union were made on July 23, 1958, April 8, 1959, and October 28, 1959. Table 1 compares the increase in size of the trees growing in the treated and untreated sites.

[1]Plant Pathologist and Associate Specialist, Department of Plant Pathology; and Assistant Biometrician, respectively, University of California Citrus Experiment Station, Riverside.
[2]Farm Advisor, San Diego County, California.
[3]Acknowledgment is made here of the fine cooperation of the owner, Mr. C. W. Ide and Mr. John Hankey, Manager of "Friendly Farm."
[4]The "American Standards Association Sectional Committee on Common Names for Pest Control Chemicals K62" has not yet assigned "coined names" for Vapam and Mylone.
[5]Trees donated by N. P. Walker Nursery of Vista, California. Samples of soil including fibrous root taken from these trees in the nursery failed to yield Phytophthora spp.

FIGURE 1. Sweet orange rootstocks of trees that failed. Note absence of fibrous feeder roots except those at top of root system which grow in soil that dries out rapidly.

FIGURE 2. A group of trees (Washington Navels on Troyer citrange rootstocks) of same age: left, tree in untreated soil; center, in soil treated with 9 ounces of 85% Mylone in basin of 50 square feet, right, in soil treated with a pint of Vapam in basin of 50 square feet.

Table 1. Effect of soil disinfestants on growth rate[a] of navel orange trees on Troyer citrange rootstocks.

Group number	Increase in circumference of trunk in millimeters[b]		
	Check	Mylone[c]	Vapam[d]
1	23	63	66
2	60	76	84
3	52	62	64
4	41	77	60
5	70	64	72
6 (Fig. 2)	9	63	72
7	24	59	59
8	36	81	73
Mean	39.4**	68.1	68.8

[a] Period of 437 days, July 23, 1958, to October 28, 1959.

[b] Measured 6 inches above bud union; marked with red Duco "touch-up" paint.

[c] Mylone W 85%, 9 ounces per basin, 50 square feet.

[d] Vapam 4S, 1 pint per basin, 50 square feet.

** The Mylone and Vapam means were significantly different from the check mean at the 0.01 level of probability.

Since the cost of either of the chemicals for disinfesting a tree site of 50 square feet is about 40 cents and labor of basining and treating is about 50 cents, the treatment is economically feasible. The average increment of growth in circumference of the trees in the disinfested sites after 437 days was 74% greater than those in untreated areas. This strongly indicates that the treatments are worthwhile under some conditions even where trees on Troyer rootstocks are to be planted. Care should be taken to prevent or delay recontamination of the disinfested sites by using uncontaminated water for irrigation, by excluding runoff water from the basins, and by dusting the basin surface with a neutral copper spray material. If the young tree grows well during its first 2 years it will, if given good subsequent cultural treatment, become a successful orchard tree. The young tree planted in soil with a high concentration of root-destroying organisms may be killed or so handicapped in its growth that it never develops into a commercially productive orchard tree.

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THE INFLUENCE OF GIBBERELLIC ACID ON SEEDLING BLIGHT OF CORN[1]

Roy D. Wilcoxson and Theodore W. Sudia[2]

The influence of gibberellic acid in promoting increases in plant height has been demonstrated by Marth et al. (3), and in some instances increases in root growth have also been reported (4). This chemical appears to have promise for use in agriculture; however, relatively little information is available regarding its influence on plant disease. It is reported to reverse virus-induced stunting (2) but aggravates root rot problems (1). The present work was undertaken to learn whether gibberellic acid might be useful in establishment of corn seedlings.

Disease severity on seedlings of inbred corn line W-10 (susceptible to seedling blight) and hybrid corn AES-202 (moderately resistant) was studied in the greenhouse during December 1958 and January 1959. Temperature of the greenhouse was approximately 65° F. There was no supplemental light.

Seeds of both corns were immersed in distilled water or solutions containing 5, 10 and 20 ppm gibberellic acid for 12 and 24 hours and planted in steamed soil. Ten ml of a mycelial suspension of Fusarium graminearum Schwabe was poured over the seeds of W-10 corn before they were covered with soil. The inoculum was poured onto the soil after the seeds of hybrid AES-202 were covered. Notes were taken about 3 1/2 weeks after planting.

Leaves of both corns from seeds treated with gibberellic acid were 1/2 to 1 inch longer, depending on the concentration of the chemical, than were leaves of plants from seeds immersed in water; however the difference was not statistically significant.

Root length of corn hybrid AES-202 was significantly increased by the chemical ($P = 0.95$). The roots of hybrid corn AES-202 were 1 and 2 inches longer, respectively, when the seeds were immersed in 10 and 20 ppm gibberellic acid than when they were immersed in water or a 5 ppm solution of the chemical. Roots of inbred corn W-10 were, on the average, 1 inch longer when the seed was immersed in gibberellic acid rather than in water; the difference was not statistically significant.

Even though the chemical appeared to stimulate the development of longer roots, it resulted in slightly higher (not statistically significantly higher) root-rot ratings than did water alone. Root rot was estimated visually on a scale of 0-4 where 0 is lowest and 4 is highest root rot.

Another indication that gibberellic acid increases severity of seedling blight was obtained in experiments on emergence and survival of seedlings. With W-10, 66% of the plants from seeds treated with 5 to 10 ppm gibberellic acid survived, but only 50% of the plants from seeds treated with 20 ppm of the chemical survived. This difference was significant ($P = 0.95$).

In these studies immersion of seeds 12 hours in the gibberellic acid solutions was just as effective as immersion for 24 hours.

An experiment was made in the field during the summer of 1959 at St. Paul, Minnesota, using Minhybrids 412, 511, 416, and 512. Seeds were immersed either in distilled water or a solution of 100 ppm of gibberellic acid for 12 hours and then treated with enough captan to thoroughly coat the seed. Corn was planted May 18, 1959 in a field on which corn had been grown for the last 15 years and which contained organisms that cause seedling blight and root rot.

Two weeks after planting, seedlings from gibberellic acid treated seeds averaged 1 1/2 inches taller than did seedlings from seeds immersed in water. Seedlings of corn seed treated with the chemical varied from 1/2 to 9 inches in height, whereas plants from seeds immersed in water were more uniform in height; the variation was not more than 1 1/2 inches from the average height. Ninety-nine% of the seeds sown produced plants in all plots.

In addition to varying greatly in height, gibberellic acid treated plants were also chlorotic and seedling blight appeared to be severe. Roots of treated plants were light brown in color and only a few adventitious roots formed. Roots of plants not treated with the chemical were white and adventitious roots were abundant (Fig. 1).

It was the opinion of those who saw the plots that the gibberellic acid treatment would result in almost complete loss of stand. During the first week of June 1959, however, the weather at St. Paul became unusually hot and dry and the treated plants recovered from their unthrifty condition. At tasseling time and thereafter the effects of gibberellic acid treatment were no

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longer apparent.

FIGURE 1. The effect of gibberellic acid on corn seedlings. The three plants on the left were from seed treated with the chemical; the three on the right were from seed soaked in water. Note the light color of foliage and the few roots on the treated plants.

Yields were obtained on October 10, 1959 from 15 plants of each hybrid and each treatment. The average weight of oven-dried seed from gibberellic acid treated plants was 3974 gm while that from the untreated plants was 4193 gm. The difference was not statistically significant.

From these studies it would appear the gibberellic acid may increase severity of corn seedling blight. Before the chemical is used in connection with commercial corn production more work should be done to fully determine its influence on disease.

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DISEASES OF FORAGE LEGUMES IN MINNESOTA[1]

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During the last 30 years forage legumes have become increasingly more important in the agriculture of Minnesota. This statement is particularly true of alfalfa which now occupies 2 1/2 million acres of land in the State. Alfalfa for forage is valued at $100,000,000 and for seed at $100,000 annually. In addition, other forage-legume crops grown in the State are valued at more than $25,000,000 annually.

Within the last 10 years, evidence has accumulated to suggest that diseases are among the important factors causing losses in forage-legume crop production in Minnesota. To confirm previous findings as to the importance of certain diseases, and to enlarge our understanding of the disease situation in general, the disease surveys reported here were begun in 1956.

Frequent surveys of growers' fields of alfalfa (Medicago sativa L.), red clover (Trifolium pratense L.), sweet clover (Melilotus spp.), and other forage legumes were made by the authors during the growing seasons of 1956 through 1959. The fields were chosen at random in all parts of the State and the diseases present were rated for severity and possible damage caused. In many instances the fungi and bacteria associated with particular symptoms in the field were isolated and identified in the laboratory. The disease information is summarized in Tables 1, 2, and 3.

Table 1. Diseases of alfalfa and their relative importance and distribution in Minnesota from 1956 to 1959.

Disease	Causal organism	Importance	Distribution	Seasonal occurrence
Spring blackstem, leaf spot	Phoma herbarum var. medicaginis (Ascochyta imperfecta)	major	general	spring, early summer, fall
Pseudoplea leaf spot, brown spot, scorch	Pseudoplea briosiana	major	general	1956, 1957, 1958 on second and succeeding hay crops; 1959, all season
Common leaf spot	Pseudopeziza medicaginis	major	general	throughout season
Bacterial wilt	Corynebacterium insidiosum	major	general	throughout season
Root and crown rot	Fusarium spp., Rhizoctonia solani, Phoma spp.	major	general	throughout season
Bacterial stem blight	Pseudomonas medicaginis	minor	southwestern Minnesota	spring and early summer
Alfalfa mosaic	Alfalfa mosaic virus complex	unknown	general	symptoms during cool seasons
Summer blackstem, Cercospora leaf spot	Cercospora zebrina	minor	southern half of State	summer, early fall
Blackstem, brown leaf spot	Colletotrichum destructivum	unknown	general	throughout season
Stemphylium leaf spot	Stemphylium botryosum	minor	general	summer and fall
Yellow leaf blotch	Pseudopeziza jonesii	minor	northwestern Minnesota	summer
Downy mildew	Peronospora trifoliorum	minor	general	throughout season
	Stagonospora meliloti	minor	unknown	unknown
Rust	Uromyces striatus var. medicaginis	minor	general	fall; varies considerably from season to season

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Table 2. Diseases of red clover and their relative importance and distribution in Minnesota from 1956 to 1959.

Disease	Causal organism	Importance	Distribution	Seasonal occurrence
Blackstem and leaf spot	Phoma herbarum var. medicaginis	major	general	mostly on older plants all summer
Northern anthracnose	Kabatiella caulivora	major	general	spring and fall; all season in north
Virus	Virus complex	major	general	throughout season
Root rots	Fusarium spp. and Pythium spp.	major	general	throughout season
Bacterial blight	Pseudomonas syringae	minor	general	throughout season
	Colletotrichum destructivum	unknown	general	throughout season
Leaf spot	Cercospora zebrina	major	general	mid-summer through fall
Target spot	Stemphylium sarcinaeforme	major	general	mid-summer through fall
Pepper spot	Pseudoplea trifolii	minor	general	mid-summer through fall
Sooty blotch	Cymadothea trifolii	minor	general	mid-summer through fall
Rust	Uromyces trifolii	minor	general	mid-summer through fall
Powdery mildew	Erysiphe polygoni	unknown	general	mid-summer through fall
Common leaf spot	Pseudopeziza trifolii	minor	general	mid-summer through fall

Table 3. Diseases of sweet clovers[a] and their relative importance and distribution in Minnesota from 1956 to 1959.

Disease	Causal organism	Importance	Distribution	Seasonal occurrence
Summer blackstem	Cercospora davisii	major	general	mid-summer through fall
Blackstem	Ascochyta meliloti	major	general	throughout season
Common leaf spot	Pseudopeziza meliloti	major	general	throughout season
Stem canker	Ascochyta caulicola	minor	general	mid-summer through fall
Leaf spot	Stemphylium botryosum	minor	general	throughout season
Leaf spot	Pseudoplea sp.	minor	general	throughout season
	Colletotrichum destructivum	unknown	general	throughout season
Virus	Virus complex	unknown	general	throughout season

[a]Melilotus alba Desr. and M. officinalis (L.) Lam.

The only destructive new disease appearing on alfalfa during this 4-year period was Pseudoplea leaf spot caused by Pseudoplea briosiana (Poll.) Hoehn. Although it had been observed in the State as early as 1952, an epidemic of the disease did not occur until 1956 when the second and succeeding hay crops were severely damaged generally over the State. In 1957 Pseudoplea leaf spot was also damaging and widespread, but in 1958 it was important only in the western portion of the State. In 1959, the disease was severe for the first time on the first hay crop from stands 2 years old or older in many parts of the State.

Colletotrichum destructivum O'Gara, the cause of brown leaf spot, was consistently isolated from alfalfa stems and leaves throughout the entire State. Although the fungus is universally present, the extent of damage it causes has not been determined. C. destructivum, as well as several other organisms, causes blackstem.

Foliar diseases of red clover are generally more important after July 1 in stands which have not been harvested. In such fields nearly all of the diseases listed in Table 2 can be found, but in some fields certain diseases are more destructive than others. Virus diseases, root rots, and northern anthracnose are probably the most important diseases of red clover in Minnesota at present.

Only a few fields of ladino and white clover, alsike clover, and birdsfoot trefoil were examined. Except for northern anthracnose, the same diseases were found on white (T. repens L.) and alsike clovers (T. hybridum L.) as were found on red clover. On birdsfoot trefoil (Lotus corniculatus L.) we have seen light infection caused by Stemphylium loti Graham, Cercospora sp. and Colletotrichum sp.

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CHEMICAL CONTROL OF PEACH TREE CHLOROSIS[1]

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Chelated iron compounds have been tested for control of peach tree chlorosis in Colorado for several years. The best responses have been obtained from soil applications. Foliage sprays have not given satisfactory results.

Previously, a pot-hole method was used in which the materials were placed proportionately in holes 8 inches in diameter and 10 inches deep at equal distances of 4 feet from the tree. Thus the material was localized with respect to the root system. Results of these tests showed favorable response to treatment. However, there was considerable variability in the response of individual trees; in some cases the foliage did not regain its normal green color uniformly. This suggested that greater recovery from chlorosis could be expected by distributing the iron chelate more evenly in the soil zone occupied by feeder roots.

In tests performed in 1958, Versenol iron chelate was applied to 10 uniformly chlorotic trees at the rate of 2 pounds per tree. Two kinds of placement were used. Five trees received short bands 6 inches wide and 12 inches long of the dry chelated iron, which is somewhat comparable to the pot-hole method. The second group of five trees received long bands 6 inches wide and 48 inches long of iron chelate. The material was applied proportionately in the bottom of two furrows, one on each side of the tree. Five trees were left as non-treated controls. Best corrective responses were obtained with the long-band method of placement. This indicated that a more uniform response is obtained by a more even distribution in the zone of active absorption. The long-band method of application was shown to be more effective than the pot-hole method.

Outstanding results were obtained in the same orchard in 1959 tests, using Sequestrene 138 Fe and 330 Fe, manufactured by the Geigy Chemical Corporation, and Versenol F, manufactured by the Dow Chemical Company. These were applied to the soil at the rate of 1 1/2 pounds per tree. The materials were applied in 14-foot bands in the irrigation furrow on each side of the tree. Where applications are described herein as band treatments, the depth of the furrow was increased about 2 inches, the chemical applied in the dry form and covered with soil to prevent it from washing away in the irrigation water. The applications were made on May 21, immediately preceding the second irrigation.

Table 1. The restorative effects of chelated iron compounds about 7 weeks after application (May 21-July 7).

	Incidence of chlorotic foliage in several severity classes[a]				
Chemical	None	Trace	Slight	Moderate	Severe
Sequestrene 138 Fe	99.8	0.2			
Sequestrene 330 Fe	66.0		10.0	24.0	
Versenol F	97.8	0.2		2.0	
Non-treated control					100

[a]Incidence of chlorotic foliage is an average reading of five trees expressed in %. Severity of chlorosis rated as follows: None, foliage dark green; trace, faint mottle on 1% of foliage; slight, faint interveinal mottle exceeding 1% of foliage; moderate, distinct interveinal mottle; severe, bright yellow leaves with necrotic margins, defoliation of terminal shoots.

As shown in Table 1, it is significant that all the trees treated with Sequestrene 138 Fe and Versenol F were almost completely free of chlorosis, whereas trees receiving Sequestrene 330 Fe showed moderate chlorosis in 24% of the foliage. The restorative effect of Sequestrene 138 Fe was somewhat more rapid than Versenol F; however, 2 weeks after the first readings were made, the differences in response were imperceptible. Sequestrene 330 Fe gave good response, but the foliage of individual trees did not recover uniformly. The improved method of placement does not seem to account for the variability in response to this material. All non-treated trees in the control developed severe chlorosis.

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FUNGICIDAL CONTROL OF MANGO ANTHRACNOSE[1]

Minoru Aragaki and Mamoru Ishii[2]

Abstract

The use of chemicals[3] has been the only feasible means of controlling mango anthracnose in all its manifestations. Using Haden and Wootten as test varieties, spray tests were conducted during 1955 and 1957. Increased Haden yield was obtained with zineb and good fruit protection was obtained with captan in the 1955 test. Increased yields and a high degree of fruit protection were obtained with all the treatments of the 1957 test; treatments B and D (Table 3) were the best treatments from the overall viewpoint.

INTRODUCTION

Anthracnose of mango is very common and widespread in Hawaii; it is the most important disease and a limiting factor in mango production in the high-rainfall areas. It is reported as the most prevalent and destructive disease of mango in Florida, the West Indies, and India (2, 8, 9).

The causal organism of mango anthracnose, Colletotrichum gloeosporioides Penz., is an ubiquitous fungus on many tropical plants. Under favorable conditions it is an important pathogen of many fruits and ornamentals. Anthracnoses of papaya, avocado, citrus, and other tropical fruits are caused by this fungus; withertip of citrus and apple bitter-rot are also caused by C. gloeosporioides.

REVIEW OF LITERATURE

Mango varieties in general have shown very little in the way of resistance to anthracnose. Pirie, a very desirable mango variety, has been found very susceptible to anthracnose (6). Haden has been observed to be more tolerant to anthracnose than most other varieties, but frequent heavy infections have occurred which have caused considerable loss. Yee (11) has recommended Paris and Fairchild as varieties which are relatively resistant.

Because of the lack of resistance to anthracnose in the desirable varieties of established orchards in Hawaii, the use of chemicals to control the disease has been necessary. Until the advent of organic fungicides about 15 years ago, copper fungicides were used exclusively. Varying levels of successes were obtained with copper fungicides such as Bordeaux mixture, Burgundy mixture, basic copper sulfate, cuprous oxide, and copper oxychloride (2, 3, 4, 5, 7, 9, 10). The main objections to the copper fungicides were those of toxicity to the open blooms and the accompanying increase in scale and mealybug infestations.

Hendrix et al. (3) tested a number of organic fungicides on several mango varieties and reported that some were promising against anthracnose. Among these were dichlone, ziram, ferbam, tris(2-hydroxyethyl)(phenylmercuri) ammonium lactate, and 2-dodecylisoquinolinium bromide.

Ruehle (7) reported on tests conducted with a number of organic fungicides and found that successful control of anthracnose can be obtained with ferbam, zineb, maneb, captan, and the copper derivative of 8-quinolinol. He found that dichlone was ineffective against anthracnose and in adddition caused a blotching of the fruit. In a later report, Ruehle and Ledin (8) recommended captan, maneb, and zineb for open bloom sprays, and copper fungicides to protect the fruit. Aragaki and Goto (1) have reported on the effectiveness of captan and zineb against anthracnose on the extremely susceptible Pirie mango.

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[3]Fungicides and adjuvants used in these tests were supplied by the following companies: Stauffer Chemical Co., San Francisco, California; E. I. du Pont de Nemours & Co., Wilmington, Delaware; Tennessee Corp., Atlanta, Georgia; Rohm & Haas Co., Philadelphia, Pennsylvania; Hawaiian Agricide and Fertilizer Co., Honolulu, Hawaii; California Spray-Chemical Corp., Richmond, California.

MATERIALS AND METHODS

The Haden mango, a popular variety which is moderately susceptible to anthracnose, and the Wootten, a high-yielding variety which is highly susceptible to anthracnose, were selected as test varieties for these series of tests. Test trees were all located in the orchards of the Poamoho Experimental Farm, North Oahu, located at an elevation of 650 feet with an average annual rainfall of 38 inches.

In all of the tests individual panicles were labeled with shipping tags for identification. Spray applications were made with a knapsack or light-power sprayer and subsequent data were collected from only these panicles.

Fruit set counts were made at intervals and fruit were harvested at the onset of color change and incubated at room temperature for several days before readings were made. All maturing fruit were harvested. Marketable fruit and cull fruit were separated on the basis of final weights. Those weighing 150 grams or approximately 1/3 pound and above were classified marketable and those less than 150 grams were classified culls.

Experiment 1. 1955 Haden Spray Test: In this preliminary spray test a number of suitable fungicides were compared. Captan, zineb, maneb, and basic copper sulfate were tested for their ability to control anthracnose. Ten trees were selected in the Haden orchard at the Poamoho Experimental Farm, North Oahu. These trees were selected for their uniformity in the development and distribution of the panicles. Each tree was treated as a replicate and divided into five sectors. Each sector represented a plot and was made up of ten tagged panicles.

The first of nine biweekly spray applications was made on April 5, 1955, at the time of open bloom. The last spray application was made nearly a month before the first fruit were harvested. The fungicides tested were captan (50% W.P. at 4 pounds/100 gallons), zineb (65% W.P. at 2 pounds/100 gallons), maneb (70% W.P. at 2 pounds/100 gallons), and basic copper sulfate (98% W.P. at 4 pounds/100 gallons). Triton B-1956 (modified phthalic glycerol alkyd resin) was added at the rate of 6 ounces per 100 gallons to each.

Fruit set counts were made at varied intervals for each tagged panicle, and fruit were harvested as the base color began to turn yellow and were kept in the laboratory for 7-day storage periods, at the end of which they were checked individually for anthracnose development. Cull fruit were harvested in the same manner but were kept for only 3 days before observations were made.

Experiment 2. 1957 Haden Spray Test: The 1955 spray test showed outstanding anthracnose control with captan and greater fruit set with zineb. Spray schedules incorporating these attributes were devised in an attempt to obtain increased fungicidal control of the withertip and fruit spot stages of the disease. Captan was used in three of the spray schedules; zineb was applied during the flowering period for two of the treatments; and cuprous oxide was applied as fruit protectant for one treatment. Table 1 outlines the spray schedules of the various treatments. Wetting Agent 60-L (60% solution of alkyl aryl sodium sulfonate) was added to each treatment at 6 ounces per 100 gallons.

Each treatment was represented by seven trees. Seventy panicles were tagged on each tree; of these, 20 panicles were marked with colored tags for fruit set counts and the remaining 50 were marked with white tags.

The first spray application for all treatments was made on April 10, 1957. The average panicle length was approximately 5 inches and there were no open blooms at the time of the first spray application. The third and fourth weeks represented the major flowering period and by the fifth week there were few flowers on the trees. At the time of the ninth week, when the last application for treatments A, B, and C was made, the fruit were approximately one-third of maturity. By the thirteenth week, when the last application for treatment D was made, the fruit were two-thirds to maturity. Fruit set counts for 20 panicles of each tree were made on the tenth and sixteenth weeks.

Forty apparently clean, marketable fruit from each tree were selected for a test of their keeping quality. The fruit were kept for a 4-day period and counts of anthracnose infection were made at the end of the fourth day. The fruit were probably a day past the best eating stage as to firmness but were otherwise quite acceptable after this period.

Experiment 3. 1957 Wootten Spray Test: The Haden variety of mango is noted for irregularity in its bearing habit (8, 11). One of the principal causes of this irregularity is undoubtedly anthracnose.

Wootten, a variety which had been observed to be a heavy bearer but at the same time to be

quite susceptible to anthracnose, was selected as the test plant in order to determine the effect of anthracnose control on a heavy-bearing variety. Two trees, each representing a replicate, were divided into three sectors of 50 tagged panicles. Treatments were captan at 3 pounds/100 gallons, zineb at 2 pounds/100 gallons, with the Wetting Agent 60-L at 6 ounces/100 gallons, and unsprayed controls. The first application was made February 7, 1957, when the panicles averaged 5 inches in length, then followed by four weekly applications to cover the open blooms with fungicide. Three additional applications were made at 3-week intervals, making a total of eight applications.

Fruit set counts were made on three separate dates; the final count was made when the first fruit were beginning to ripen. No harvest data were taken for this test.

EXPERIMENTAL RESULTS

A summary of yield and incidence of anthracnose of Experiment 1 is given in Table 2. Zineb was significantly better than basic copper sulfate in the initial fruit set (1% probability level), but there were no differences among the treatments as to their effect on final yield. Basic copper sulfate treatment resulted in the lowest initial fruit set which represents manifestation of phytotoxicity on the open bloom and is in accordance with the observations of Ruehle (7).

Although there were no significant differences among the fungicides, they were all effective in controlling the fruit-spot phase. Captan was extremely promising in this respect.

Table 3 summarizes the fruit set on the different treatments of Experiment 2. Fruit set on treatment C was lower than for the other fungicidal treatments, but not significantly. This lowered fruit set was taken to be an indication of toxicity to the open bloom by captan, which was applied weekly for this treatment during the flowering period.

Final yields for treatments A, B, and D were significantly higher (using analysis by group comparisons) than that of unsprayed controls. Treatment C, although showing a higher yield, was not significantly better than E because of the great variations between individual trees. Unsprayed fruit of treatment E were considerably larger than the others because of the lower numerical yield.

A summary of the incidence of anthracnose in the 1957 spray test is given in Table 4. Schedule D, in which spray applications were extended over a long period, and schedule B provided the best protection for the fruit, although schedule C was nearly as good (Fig. 1).

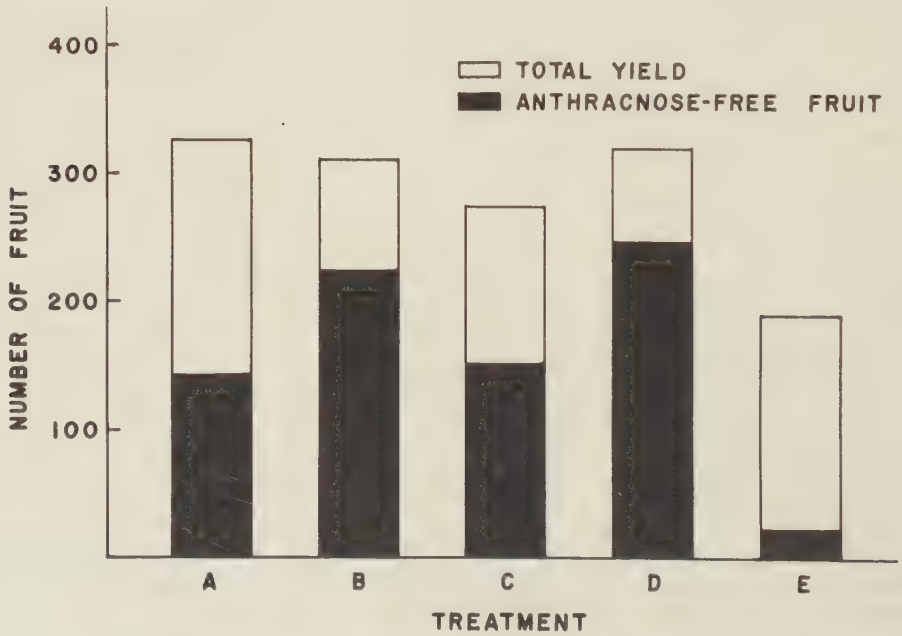

FIGURE 1. Total yield in numbers of fruit and relative proportions of disease-free fruit (1957 Haden test).

Table 1. Spray schedules (1957 Haden test).

Treatment	Week number												
	1	2	3	4	5	6	7	8	9	10	11	12	13
A	Z[a]	Z	Z	Z	Cu	-	Cu	-	Cu	-	-	-	-
B	C	C	Z	Z	C	-	C	-	C	-	-	-	-
C	C	C	C	C	C	-	C	-	C	-	-	-	-
D	C	-	C	-	C	-	C	-	C	-	C	-	C
E	-	-	-	-	-	-	-	-	-	-	-	-	-

[a]Z = zineb (65% W.P.) at 2 pounds/100 gallons. Cu = cuprous oxide (90% W.P.) at 2 pounds/100 gallons. C = captan (50% W.P.) at 3 pounds/100 gallons.

Table 2. Incidence of anthracnose (7 days after harvesting).

Fruit class	Treatment						
	Captan	Zineb	Maneb	Basic copper	Control	LSD .05	LSD .01
Marketable							
Total fruit harvested	36	49	36	36	26		
No. disease-free fruit	29	30	28	28	6		
% disease-free fruit	83	61	78	78	23		
Av. no. spots per replication	0.2	3.9	0.5	0.9	12.4	5.4	7.2
Culls							
Total fruit harvested	16	26	12	25	18		
No. disease-free fruit	9	13	6	13	0		
% disease-free fruit	69	50	50	52	0		
Av. no. spots per replication	0.1	6.0	5.3	1.3	22.0	10.3	13.8

Table 3. Fruit set and harvest data (1957).

Treatment	Fruit set[a]				Harvest[b]		
	10th week		16th week		No. fruit harvested	Weight of fruit (in pounds)	Average weight of fruit
	Total	Av.	Total	Av.			
A	192	27.4	125	17.9	327	289	0.89
B	200	28.6	127	18.1	311	296	0.95
C	144	20.6	86	12.3	236[c]	197[c]	0.83
D	186	26.6	131	18.7	320	285	0.89
E	116	16.6	64	9.1	189	189	1.00
LSD .05				7.1			
LSD .01				9.6			

[a]Counts based on 20 panicles from each of 7 trees, or 140 panicles.
[b]Yields based on 70 panicles from each of 7 trees, or 490 panicles.
[c]One tree lost in storm, total of 6 trees.

Table 4. Incidence of anthracnose (1957).

	Spray schedule					LSD	
	A	B	C	D	E	.05	.01
No. fruit examined	280	280	240[a]	280	280		
No. of spot-free fruit	123	202	133[a]	217	37		
Av. no. of spot-free fruit	17.6	28.9	22.2	31.0	5.3	8.1	11.0
No. of spots	1352	342	529	199	5030		
Av. no. of spots	193.1	48.9	75.6	28.4	718.6	80.7[b]	109.5[b]
No. of spots per diseased fruit	8.6	4.4	4.9	3.2	20.7		

[a]Six-tree total.
[b]Treatment E not included in analysis.

Table 5. Set of Haden, based on harvested fruit.

Year	Treatment	No. panicles	No. fruit	Fruit per panicle
1955	sprayed	400	157	0.39
	unsprayed	100	26	0.26
1957	sprayed	1890	1194	0.63
	unsprayed	490	189	0.39
Total	sprayed	2290	1351	0.59
	unsprayed	590	215	0.36

Table 6. Fruit set[a] of Wootten (1957).

Treatment	April 24	May 22	June 19
Captan	371	145	113
Zineb	186	128	90
Unsprayed	71	39	29

[a]100-panicle total.

Cuprous oxide, which was used to protect the fruit in schedule A, was significantly poorer than captan used in the other schedules. Not only were there more diseased fruit, but there were twice as many spots per infected fruit in schedule A. On the basis of yield and incidence of fruit spot, schedules B and D were the preferred treatments.

During the course of this investigation, fruit data were collected on the basis of hundreds of tagged Haden panicles. Table 5 gives a compilation of marketable Haden fruit harvested from sprayed and unsprayed panicles. Increased yield in marketable fruit of sprayed trees over unsprayed trees were 50% in the 1955 test and 61% in the 1957 test. The higher gain in yield for the 1957 test is attributed to the higher incidence of anthracnose on marketable fruit during the 1957 test.

Table 6 summarizes the results of Experiment 3. Captan sprayed weekly during the open bloom did not seem to lower the set of Wootten fruit, as it did in the variety Haden. In fact, an extremely high initial set was obtained with captan, which was gradually lost, and at harvest time there was little to choose between captan and zineb, but both were significantly better than the unsprayed control.

The increased fruit set with captan may have been due to a higher captan tolerance by Wootten or to better disease control of a highly susceptible variety by captan. The high degree of anthracnose control during the open bloom period seemed to overshadow the toxic effects of captan. This effect of captan on a highly susceptible variety was noticed but not recorded by the senior author in a previous report on the mango variety Pirie (1).

Phytotoxicity of Captan

Ruehle (7) and Ruehle and Ledin (8) reported that captan was incompatible with liquid spreaders such as Triton B-1956 and recommended the use of dry spreaders with captan.

In our 1955 Haden spray test, numerous tiny red scabs were noticed on captan-sprayed fruit at harvest time. These scabs, not more than 2 mm in diameter and irregular in outline, were invariably associated with the lenticels. Because of their inconspicuousness, they were not noticed until the last spray application was being made and, in the opinion of the authors, they did not detract from the attractiveness of the fruit. In this test the proprietary adjuvant Triton B-1956 was used at the rate of 6 ounces per 100 gallons and the last spray application was 9 weeks before the first fruit were harvested.

In the 1957 test another liquid proprietary adjuvant, Wetting Agent 60-L, was used at the rate of 6 ounces per 100 gallons. In this test no fruit abnormalities as described above were noticed with the use of captan.

Since the two adjuvants just mentioned were not used under comparable conditions, a comparison of the two was made in 1958. The flowering was early but very sparse that year so that comparisons could be made on different sectors of a single tree. Captan at the rate of 3 pounds per 100 gallons was sprayed with the adjuvants Triton B-1956 and Wetting Agent 60-L, each at 6 ounces per 100 gallons. Schedule D of the 1957 test (Table 3) was followed; the seventh and last application was made on May 13, 1958, more than 2 months before the first fruit ripened.

No scab, as noted in the 1955 test, nor injury in any other form was discernible in this

test with captan and either of the adjuvants mentioned.

Neither the nature of the scab nor the conditions under which it occurs is clear at this time, although it appears that spray applications made during the last few weeks may be the causal factor. At any rate, the injury is innocuous and has appeared in our experimental plots only once out of three tests made.

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RIO OSO GEM PEACH SEEDLINGS AS INDICATOR HOSTS FOR THE PRUNUS RING SPOT VIRUS[1]

T. S. Pine[2] and Harold E. Williams[3]

Abstract

Rio Oso Gem peach seedlings and Shiro-fugen cherry trees were simultaneously inoculated with cultures of the Prunus ring spot virus. Sixty-three of the 68 cultures tested gave positive readings on both indicator hosts. The remaining five cultures gave mixed reactions on both hosts. It was concluded that Rio Oso Gem seedlings and Shiro-fugen trees were consistent in their expression of Prunus ring spot symptoms but that very mild strains of the virus may not produce symptoms on either host.

The term "Prunus ring spot virus" is used herein to refer to the virus or viruses which cause the peach ring spot virus reaction in peach as described by Cochran and Hutchins (2) or the localized necrotic lesion reaction in Shiro-fugen flowering cherry as described by Milbrath and Zeller (7). The most common method of indexing Prunus plant material for the presence of the Prunus ring spot virus in California and other western States is to place buds of the questionable trees in Prunus serrulata var. Shiro-fugen. Because Shiro-fugen does not receive sufficient winter chilling to make good growth in southern California, the standard procedure has been to place buds from suspect trees in indicator-variety index trees in the field and examine them for disease symptoms the following spring. This requires the use of valuable field space and a minimum time of 6 months before the indexing is completed. A more rapid and flexible technique was obviously needed to facilitate work with this virus. Use of peach trees for rapid tests for certain stone fruit viruses has been reported by Hildebrand (5, 6).

Seedlings of Prunus persica var. Rio Oso Gem were selected as potential test trees because the variety previously had been shown to be severely injured when inoculated with the peach ring spot virus (3), and seedlings of this variety are fairly uniform in growth characters. Test trees were obtained by planting Rio Oso Gem seeds in nursery rows and allowing the seedlings to grow in place during their first season. After reaching dormancy, the seedlings were undercut and removed from the ground and their shoots and roots were trimmed for uniformity in handling. The seedlings were made into bundles of about 60 seedlings each, placed in containers with moist peat moss and sand, and stored in a cold room at 34° F for at least 60 days.

The test seedlings were removed from the cold room and planted in 6-inch pots in the greenhouse as needed. As soon as the green-bud stage was reached, inoculum composed of two bark chips from material suspected of carrying the Prunus ring spot virus were placed in each test seedling. Symptoms of Prunus ring spot became evident in the test plants within 7 to 21 days.

A small amount of seed transmission of Prunus ring spot virus is known to occur in peach (1). Experience has shown that contaminated seedlings can be avoided by selecting from the nursery row only trees which are uniform in vigor. It has been observed that seedlings infected by seed transmission do not show bud-break until several days after healthy seedlings have begun to grow vigorously. Therefore, the seedlings used were those which uniformly showed bud-break 4 to 5 days after being brought from the cold room into the greenhouse. No instance of test failure was attributed to seedling contamination with Prunus ring spot virus in more than 1000 indexes at Riverside.

This technique was used with apparent success for several years, but the question persisted that perhaps the milder strains of the virus were not being detected. In order to gain confidence in the reliability of this procedure, tests were made with a series of cultures of Prunus

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[1]Cooperative work of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, the California Agricultural Experiment Station, and the California Department of Agriculture.

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Work contributed by this author was done under Agricultural Marketing Service Project No. SDA-Calif. -A-2, for which State funds were matched with Federal funds received from the Agricultural Marketing Service, United States Department of Agriculture, under provisions of the Agricultural Marketing Act of 1946.

ring spot virus representing strains of the virus ranging in severity from mild to severe. During the growing season of 1959, each culture was tested in Shiro-fugen flowering cherry and Rio Oso Gem seedlings which had been handled in the above manner.

Sixty-eight previously tested stone fruit virus cultures, each believed to contain a strain of Prunus ring spot virus, were obtained from widely separated locations in the United States[4]. Cultures received from locations where quarantines restrict the movement of peach wood were received in cherry. The cultures in budsticks were received at Sacramento, California, where half of each budstick was used to inoculate Shiro-fugen trees growing in a California Department of Agriculture test plot. The other half of each budstick was forwarded to Riverside, where it was used to inoculate the Rio Oso Gem peach seedlings growing in a greenhouse at the Citrus Experiment Station. Tests were made by placing two buds of each virus source in Shiro-fugen trees and two bark chips on each of two peach seedlings. The Shiro-fugen and Rio Oso Gem seedling indicator hosts were examined for disease symptoms at the ends of 30 and 21 days, respectively.

Sixty-three of the 68 cultures gave positive readings on Shiro-fugen trees and Rio Oso Gem seedlings. Three of the remaining five cultures were mildly positive on Shiro-fugen and negative on Rio Oso Gem peach seedlings, and conversely two cultures were negative on Shiro-fugen and mildly positive on the Rio Oso Gem seedlings. Budwood collected from healthy check source trees in Washington and California was uniformly negative when tested on both indicators. It therefore seems evident that Rio Oso Gem peach seedling trees, used under the conditions given above, are as consistent in their expression of Prunus ring spot symptoms as is Shiro-fugen. It also appears that very mild strains of the virus may not produce symptoms on either host but that this failure is not substantially greater with one than the other.

This method of using Rio Oso Gem peach seedlings as indicator hosts for Prunus ring spot virus has an advantage in areas where Shiro-fugen cannot be grown and may also have some advantages even in areas where Shiro-fugen grows well. The peach seedlings can be ready for use by January 1 or kept in storage at 34° F until mid-July. Thus, they are available as test plants over a period of 6 months. In plot-grown trees Prunus ring spot virus generally causes symptoms only on foliage produced early in the first growing season, or once in 12 months, whereas in Rio Oso Gem seedlings in the greenhouse, symptoms can be produced in 3 weeks. The Rio Oso Gem seedlings are available for use after field conditions preclude further symptom expression. Excessive greenhouse space is not needed for tests of large numbers of suspect trees because test plants can be discarded after 3 weeks. This technique has an advantage over the excised-twig method (4) in that symptom expression is better and the severity of different virus strains is more readily distinguishable. Symptom expressions caused by the cultures tested in the present study ranged in severity on peach from abundant leaf necrosis and terminal dieback to only occasional obscure ring patterns in the leaves.

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[4]The authors are indebted to the following persons for supplying the out-of-state virus cultures: Donald Cation, J. A. Milbrath, J. Duain Moore, K. G. Parker, and E. L. Reeves.

TWISTED LEAF AND RING POX VIRUSES FOUND IN CHOKECHERRIES NEAR DISEASED ORCHARDS[1]

T. B. Lott and F. W. L. Keane[2]

It has been reported that chokecherry (Prunus virginiana L. var. demissa (Torr. & Grey) Torr.) is capable of carrying the twisted leaf virus without showing symptoms (1). It has also been reported that a pronounced degree of association exists between twisted leaf of cherry and ring pox of apricot (2). For these reasons an attempt was made to determine whether a virus capable of producing either disease could be transmitted out of wild chokecherry trees in the Okanagan and Similkameen Valleys of British Columbia.

MATERIALS AND METHODS

In 1957 material was collected from 244 chokecherry trees in 12 locations, all adjacent to twisted leaf or ring pox in orchard trees. In each location the chokecherries were grouped. The number of trees in a group ranged from 4 to 98, depending on the availability of native trees. The ten smallest groups were each indexed by inoculation into one young Bing cherry tree and one young Wenatchee apricot tree. The two largest groups, of 98 and 54 chokecherries, collected on different sides of the same orchard, were indexed separately in two young Wenatchee trees and together in a single larger Bing tree.

RESULTS

The results of the inoculations are presented in Table 1. The larger Bing cherry tree, which received inoculum from 152 chokecherries, was infected with twisted leaf the following

Table 1. Results of indexing wild chokecherries by inoculation into Bing cherry and Wenatchee apricot. 1957-1959.

Collection and location	Number of chokecherries	Adjacent to twisted leaf or ring pox	Disease in Bing test tree and year	Disease in Wenatchee test tree and year
Okanagan Valley				
1. Summerland	13	both	none	none
2. do.	4	both	none	none
3. do.	98	both	1958 twisted leaf	1958 - ring pox in fruit
4. do.	54	both		1958 - ring pox in fruit
5. do.	15	both	1958 twisted leaf	
6. Penticton	9	ring pox	none	none
7. do.	4	ring pox	none	none
8. do.	7	ring pox	none	none
9. do.	7	ring pox	none	none
10. do.	11	ring pox	none	none
11. Osoyoos	6	ring pox	none	none
Similkameen Valley				
12. Cawston	16	both	1958 twisted leaf	1959 - ring pox leaf and twig symptoms

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spring. In this tree there appeared to be at least fifteen separate infections related to inoculations, while uninoculated parts of the tree still appeared normal. However, the group indexing used in this work could not determine the virus content of any individual chokecherry or the number of infected chokecherries in any group.

Twisted leaf was obtained out of three groups of symptomless wild chokecherries, two in the Okanagan Valley and one in the Similkameen Valley.

Ring pox was also obtained out of three groups of symptomless wild chokecherries, two in the Okanagan Valley and one in the Similkameen Valley, with diagnosis of the last depending on leaf and twig symptoms only, in the absence of fruit.

In two cases, one in each valley, twisted leaf and ring pox were obtained out of the same group of symptomless chokecherries. It was not demonstrated that the two diseases were obtained out of the same individual wild tree.

DISCUSSION

This work demonstrated that chokecherry is a host of the virus of apricot ring pox, and that infected chokecherry trees can be symptomless.

It was also found that the virus causing twisted leaf in sweet cherry and the virus causing ring pox in apricot are now present in symptomless chokecherry in both the Okanagan and Similkameen Valleys, and in the same groups of wild trees though not necessarily in the same individual trees.

This work was limited to locations close to ring pox or both twisted leaf and ring pox in orchard trees.

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TWISTED LEAF VIRUS INDIGENOUS IN CHOKECHERRY[1]

T. B. Lott and F. W. L. Keane[2]

It has been reported that both twisted leaf of cherry and ring pox of apricot were obtained by transmission out of symptomless trees of the wild chokecherry (Prunus virginiana L. var. demissa (Torr. & Grey) Torr.) growing close to diseased orchard trees (2). This work did not show whether natural spread, if any, had been into or out of the orchards. In 1958 an attempt was made to determine whether the twisted leaf virus was indigenous in the wild chokecherry. The corresponding work on ring pox of apricot was not possible until 1959.

MATERIALS AND METHODS

In the late summer of 1958 material was collected from 1471 chokecherries in 69 locations in and around the Okanagan and Similkameen Valleys of British Columbia. An attempt was made to sample native chokecherries uninfluenced by orchards. Collections were made on the floors of the valleys and well up on their partly wooded sides, and along roads through the hills between the two valleys and leading away. A single collection was made north of the Columbia watershed on the Fraser watershed. The area in which collections were made extended 100 miles north from the International Boundary and 35 miles from east to west.

Collections were made in selected locations. The number of chokecherries from which material was collected was dependent upon local conditions. Some chokecherries were large old individual trees and others were small stunted bushes. Quite often they grew in thickets so that apparently individual trees might be either clonal suckers or seedlings. Clonally related material was not wanted but was probably obtained to some extent.

Seedlings of Van cherry were inoculated with chokecherry and budded with five buds of healthy Bing in 1958. These seedlings, 3 to 5 feet high, were inoculated so that one seedling was used to index all the chokecherries collected in one location. Single cherry seedlings received up to 50 small inoculum pieces, each piece from a different chokecherry.

RESULTS

Observations were made on the inoculated trees in the spring of 1959. Some of the Bing buds failed to grow. Much of this bud failure was evidently due to the inoculations and some of the seedlings themselves showed very severe twisted leaf. However, for the sake of clarity it was decided to present only those results which were obtained in Bing. Table 1 and Figure 1 present the results of inoculations made with material collected from 1018 chokecherries in 49 locations.

Definite twisted leaf in inoculated Bing cherry trees showed that the twisted leaf virus was present in 34 locations where material had been collected from 774 chokecherries. Absence of the virus is so far indicated in 15 locations where material was collected from 244 chokecherries. These negative results are not conclusive because symptoms of twisted leaf sometimes fail to appear until the second year. The presence of the virus was demonstrated in wild chokecherry in 16 locations at least 1 mile away from the nearest orchard, and in one location 19 miles from the nearest orchard. The virus was also found in chokecherry at an elevation of 1900 feet above the nearest orchard. It was also shown to be present in wild chokecherry in 21 locations at least 2 miles away from the nearest known twisted leaf in sweet cherry or ring pox in apricot. In one instance the distance was 25 miles.

DISCUSSION

The authors consider that these results show that the twisted leaf virus is both indigenous and generally present in wild chokecherries in and around the Okanagan and Similkameen Valleys in British Columbia. Presence of the virus in a wild plant may explain the appearance of twisted leaf in orchard trees where no source of infection has been apparent.

There is almost a certainty that the virus of apricot ring pox is similarly indigenous and generally present in wild chokecherries in these same areas. This probability is indicated by the simultaneous consideration of the present work, of the demonstrated association of twisted leaf in cherry and ring pox in apricot (1), and of the obtaining of both diseases out of chokecherries close to orchards (2).

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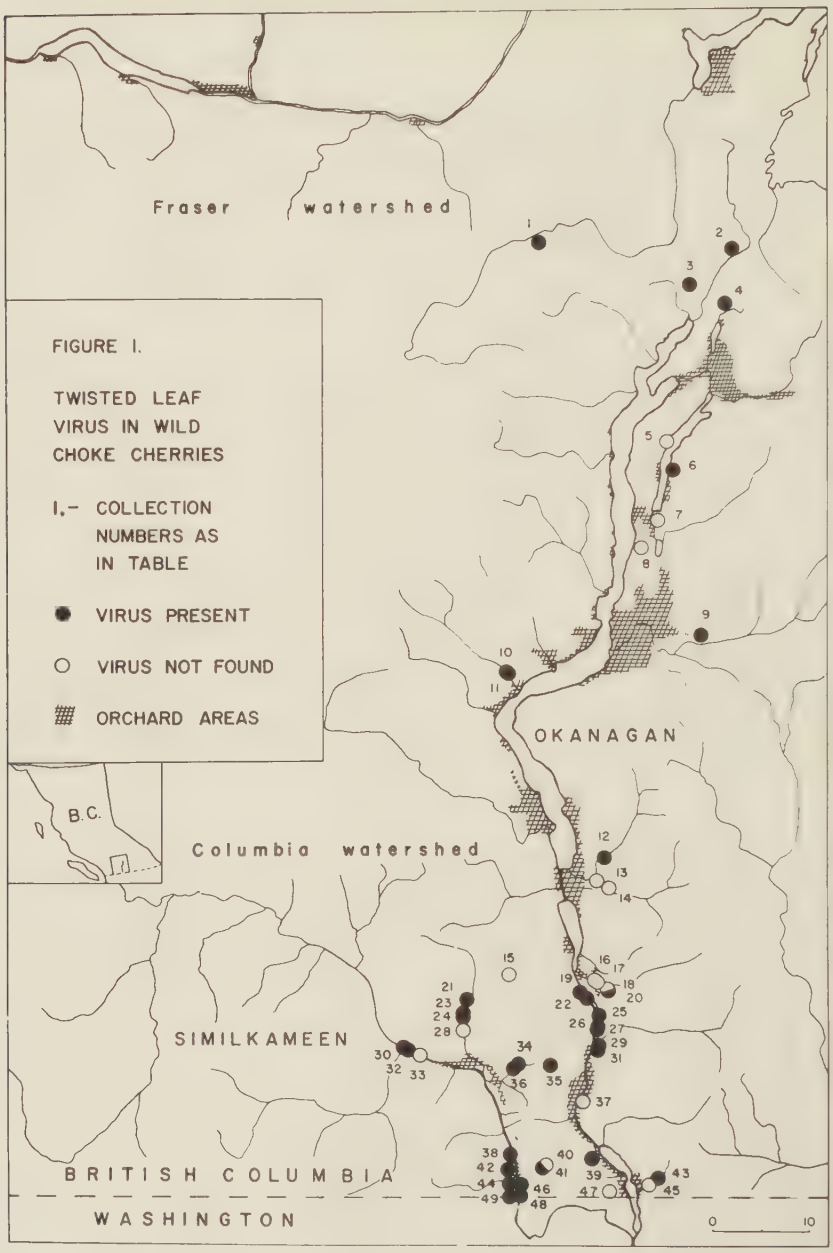
Table 1. Results of indexing wild chokecherries for twisted leaf virus by inoculation into Bing cherry.

Location	Distance from International Border (in miles)	From nearest orchard		Distance from nearest twisted leaf or ring pox (in miles)	Number of choke-cherry trees	Twisted leaf virus in choke-cherry
		Distance (in miles)	Elevation (in feet)			
1	101.0	19.1		25.0	24	present
2	100.0	1.2		16.0	18	present
3	96.4	5.0		12.6	25	present
4	94.3	1.8		10.3	20	present
5	79.9	1.5		2.3	26	absent
6	76.9	.5		1.4	21	present
7	71.4	.5		1.5	18	absent
8	68.6	1.3		3.9	10	absent
9	59.3	1.6	400	3.9	34	present
10	55.6	.6		1.8	24	present
11	55.5	.3		1.5	23	present
12	35.9	2.0	800	2.6	20	present
13	33.5	.5	500	2.5	21	absent
14	32.6	1.9	1400	3.3	2	absent
15	23.6	5.2	500	10.6	10	absent
16	22.8	.5		.5	23	absent
17	22.6	1.0	100	.7	10	absent
18	21.8	1.1	800	1.4	27	absent
19	21.7	.3		1.1	30	present
20	21.5	1.1	700	1.4	25	present
21	20.8	.2	-100	6.7	14	present
22	20.8	1.0		1.3	20	present
23	19.4	.6		5.3	8	present
24	19.1	.9		4.9	21	present
25	19.0	.3		.6	12	present
26	17.9	.3		1.7	23	present
27	17.6	.5		1.9	34	present
28	16.9	1.5	-200	3.0	7	absent
29	16.0	.9	100	3.5	26	present
30	15.8	2.0	200	3.0	23	present
31	15.6	.7		3.9	51	present
32	15.5	1.5	200	2.5	27	present
33	15.2	.5		1.3	1	absent
34	14.0	1.7	800	4.1	14	present
35	13.8	2.9	1400	3.6	37	present
36	13.5	1.1	600	3.5	7	present
37	9.9	.3	200	1.0	27	absent
38	4.6	1.2		2.3	15	present
39	4.2	.6	500	.7	31	present
40	3.3	2.7	400	2.7	4	absent
41	2.9	2.4	400	2.4	10	present
42	2.8	.7		1.2	26	present
43	2.1	1.7	1900	2.5	50	present
44	1.4	.3		1.4	22	present
45	1.3	.8	500	1.3	30	absent
46	1.2	.6		1.1	8	present
47	.8	.6	300	.8	28	absent
48	.1	.3		2.2	19	present
49	0.0	.9		2.6	12	present



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SOME SEEDLINGS OF THE VAN CHERRY FOUND TO BE SUPERIOR TO BING AS INDICATORS FOR THE TWISTED LEAF VIRUS[1]

T. B. Lott and F. W. L. Keane[2]

The Bing variety of sweet cherry has been generally used as the standard indicator for twisted leaf virus. It was known that infected Bing trees could have different degrees of severity of symptom expression. However, it has now been demonstrated at Summerland that infected Bing can be symptomless. It has also been observed that some seedlings of the Van variety of sweet cherry can show twisted leaf symptoms while Bing on the same tree remains symptomless. These observations were incidental to other work, no experiments having been made specifically for this purpose.

For several years mazzard rootstocks for virus work at Summerland have been grown from seed collected from trees of the Van variety. These Van trees were used because they had been indexed and found free from ring spot, prune dwarf, sour cherry yellows, twisted leaf expressed in Bing, mottle leaf, rasp leaf, Lambert mottle, and apricot ring pox.

The seedlings were budded with different varieties of sweet cherry and inoculated with several viruses, particularly with various selections of twisted leaf, Lambert mottle, and apricot ring pox.

In hundreds of cases a normal bud of the Bing variety was inserted into a seedling and the resulting Bing growth was used to provide symptoms of twisted leaf when that virus was present. The Van seedlings varied widely in the severity of twisted leaf symptoms which they exhibited. Usually the Bing growth was more severely affected than the seedling but some Van seedlings were more severely affected than Bing, which is the standard indicator for twisted leaf. In a few cases the Van seedling showed very pronounced symptoms with severe stunting, while the Bing showed slight symptoms, or occasionally, no symptoms at all (Fig. 1).

FIGURE 1. Seedling of Van cherry, more severely affected by twisted leaf virus than growths from inserted buds of Bing.

It is now apparent that the twisted leaf virus cannot always be detected by using Bing as the standard indicator. Thus this virus may be more widespread, without symptoms, than has been supposed. It is also apparent that certain Van seedlings can be used to detect this virus when it cannot be detected by Bing.

Those Van seedlings which have been found to be more sensitive than Bing have so far only been detected after they had been inoculated and infected. Work is under way to select uncontaminated stock of one or more such seedlings for experimental work.

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LIQUID NABAM AND N-DURE AS SUBSTITUTES FOR FORMALDEHYDE IN THE CONTROL OF ONION SMUT, UROCYSTIS CEPULAE

A. G. Newhall and R. E. Wilkinson

Liquids or powders applied in the furrow with the seed have been employed in various ways for several years. The oldest treatment with a 1% formaldehyde solution at 125 gallons per acre is still preferred by many muckland onion growers who are highly mechanized and sow six rows at a time. To the formaldehyde solution a pint of insecticide can be added to control maggots; such as aldrin, Dieldrin, parathion or 2 quarts of diazinon in areas where the maggot has become resistant to the hydrocarbons. Recently two other liquid fungicides have given as good smut control at the same concentration and rate of application as formaldehyde. These are nabam, without zinc sulfate, and N-dure, a liquid containing 59% formaldehyde and 26% urea. (U F Concentrate - 85)[1].

The chief advantages of nabam are that 1) most onion growers have it on hand all the time, as it has been used extensively for blast and mildew control for the past 7 years; 2) it is not as hard on the skin of the operator as formaldehyde; 3) it never comes in a glass returnable container on which a deposit may be required; 4) it does not have to be protected so carefully against freezing; 5) its price range is similar to formaldehyde; and 6) it has not caused injury in greenhouse trials at two or three times the 1%-concentration recommended. N-dure is a more concentrated formaldehyde solution with a "built-in" nitrogen fertilizer which theoretically can disinfest and at the same time fertilize with nitrogen, the substance most often lacking in a cold soil in April. However, the amount of nitrogen in 1 gallon of N-dure spread over 7 miles of row is negligible; in addition, conversion of urea to ammonia is slow and the ammonia must be transformed into nitrate before the plant can use it. The chief disadvantage of N-dure is its high viscosity which, at temperatures prevailing in April, makes it hard to pour; however, dilution with water will eliminate this difficulty. Theoretically, a .63% solution should be the equivalent of a 1% formaldehyde solution.

In greenhouse trials smut was reduced from a mean of 52% in the six checks to means of 2.1, 1.9, and 2.9% for nabam, N-dure, and formaldehyde respectively. A few field trials were then conducted over the State. In two of these farmers applied 125 gallons per acre of a 1% dilution of each liquid in half-acre strips across their fields. Records of seedling stands, smut percentages and yields were obtained in 1959. Smut control and yield increases over the checks were very good with all three fungicides (Table 1).

Table 1. Onion smut control with formaldehyde, nabam, and N-dure on two muckland farms in Oswego County, New York.

Treatment (125 gallons/acre)	Seedlings per 10 feet		% smutted		Bushels per acre		% increase	
	c	z	c	z	c	z	c	z
Check	80	86	43	31	724	968	-	-
N-dure, 1%	100	95	.3	1.6	882	1043	22	8
Nabam, 1%	115	81	.3	2.0	961	1080	32	12
Formaldehyde, 1%	114	89	.5	2.2	903	1163	24	20

Dithane D-14 was used for the field experiments, but greenhouse comparisons have shown that Parzate gives the same results under the same conditions. Nabam and N-dure both appear to be compatible with the insecticides currently being used for maggot control at the 1% dilutions employed in these experiments. Nabam is being recommended for grower trials on New York mucklands.

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[1]Product of Nitrogen Division, Allied Chemical and Dye Corporation, New York.

CORKY ROOT ROT OF ICEBERG LETTUCE ON THE MUCKLANDS OF NEW YORK

John K. Hoff and A. G. Newhall

Summary

A severe root rot of head lettuce grown on the acid mucklands of New York and hitherto called "stunt" or "Pythium stunt," but perhaps better designated as corky rot, has been reproduced in sand culture and in steamed muck by an excess of ammonium. A pH above 5.5 seems to reduce the severity of the injury. Symptoms in sand culture occurred with applications of ammoniated ammonium nitrate in quantities above 100 pounds of N per acre. Greenhouse tests in steamed muck, low in NH_4, have shown that corky root rot can be produced by ammoniated ammonium nitrate, urea, Uramite M, and sodium nitrite, but not by sodium or ammonium nitrate. The role played by nitrites is not yet clear, but the symptoms differ slightly from NH_4 injury. A method of quantitative determination of NH_4 in muck soil has been a great aid in correlating epiphytotic conditions with the ammonium present in a given field plot. Quantities up to 300 pounds per acre may be tolerated if pH is above 5.5, but fields where the disease is the worst have been found to contain more than 400 pounds and a pH close to 5, while fields with the least disease had less than 50 pounds and a pH above 5.4. The variety 456 is very susceptible while Big Boston, Cos and some others are resistant. In greenhouse tests over several years no typical symptoms have been produced with any of the strains of Pythium or other fungi isolated from diseased roots.

A serious root rot of head lettuce has caused losses up to 100% on some of the mucklands of New York, particularly in Oswego County where two or three lettuce crops often are grown in succession. The symptoms are identical with those pictured by Grogan and Zink (2) and described by Hannon (3, 4). Brown scurfy lesions may sometimes be found on tap roots of small plants by the time they are in the 6-leaf stage; later the entire tap root may be rotted off (Fig. 1A). The principal symptoms above ground are wilting on hot dry days and, in severe cases, some stunting. Sometimes slight pallor and, in older leaves, marginal necrosis with interveinal necrotic speckling are evident. Below ground typical symptoms are dark brown lesions and corky ridges on the tap root, rotting off of side roots, and frequently a vascular discoloration of the stele. In extreme cases the tap root becomes hollow and very dark brown inside. The name corky root is proposed here for the first time.

Many organisms have been isolated from the margins of lesions and from the discolored vascular region. The ones obtained most frequently are bacteria and species of Pythium, Fusarium, Cephalosporium, and Thielaviopsis. The discolored vascular region often yields no organisms. The frequency of any one organism varies with the time of year and field, but no typical symptoms of disease have been produced by inoculations with any of them by Hannon (3), by the junior author, or by others at Cornell[1]. Thus pathogenic organisms, although not entirely ruled out are nevertheless now believed to be of secondary importance.

Pythium wilt of lettuce is known to have occurred on the mucklands of New York 30 years ago when butter-head varieties were commonly grown; however, such varieties are tolerant to the present disease. Moreover, no outer root lesions were described in Pythium wilt (1), whereas such corky lesions are very characteristic in the present syndrome. A root rot similar to corky root was found by Thomas (8) on Grand Rapids lettuce in Ohio greenhouses; he attributed the disease to a bacterium and called the malady rosette. The facts that a rosette symptom is lacking in the head lettuce varieties grown in New York, that the brown discolored vascular region in which the bacteria were regularly found often does not yield any organism, and that corky root symptoms are induced rather than reduced by autoclaving muck soil all have led us to the belief that the two diseases are distinct.

Marlatt (5), in Arizona, described a brown discoloration of the vascular system in roots of head lettuce, presumably of the crisp head or iceberg type. The discoloration was associated with heavy applications of steer manure at 40 tons per acre. These symptoms were absent when calcium nitrate was used as the source of nitrogen. Four bacterial isolates obtained from infected roots all failed to produce symptoms in controlled experiments. Grogan and Zink (2), in experiments with head lettuce on alkaline mineral soil in California, observed

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[1]Grateful acknowledgment is made of the assistance of Dr. Grover Sowell during the summer months of 1958.

symptoms very similar to those found on New York mucklands in plants given applications of fertilizers containing ammonia in such forms as urea or ammoniated ammonium nitrate. They concluded that ammonia or nitrite injury may account for more root rotting of lettuce than has hitherto been suspected. In this the writers now concur.

THE CASE AGAINST A PATHOGEN AS THE CAUSE OF CORKY ROOT ROT

Several things have discouraged the belief that a soil organism is the primary cause of corky root rot. For example, many attempts to obtain the disease by growing lettuce in muck brought to the greenhouse from fields where crop losses were severe have failed repeatedly, even when the soil was reinforced with roots from diseased plants. As already mentioned, infesting steamed muck with pure cultures of the fungi isolated from diseased plants produced no symptoms. Moreover in field plots on several farms the use of chemical fungicides, such as formaldehyde drench, vapam, mylone, methyl bromide under cover, zinc oxide, thiram, Thylate, PCNB, Phaltan and several coded proprietary compounds has given very little control, or at best very erratic results, over a 2-year period.

The first good symptoms obtained experimentally were on the roots of young head lettuce plants grown in glass-sided boxes and 1-quart jars of muck placed under an intermittent mist in the greenhouse, which kept the soil cool and saturated, over a 3-week period similar to field conditions in the spring. This treatment raised the ammonium-N content at the expense of nitrate; ammonium-N and nitrites have long been known to be toxic to the roots of many plants.

1. Injury of Potted Lettuce Plants Fed Fertilizers Containing Nitrogen from Different Sources:

After washing and airing to eliminate excess ammonium, autoclaved muck was used in a test to determine the relative toxicity of six nitrogen fertilizers to lettuce roots. Seedlings of Imperial 456 were then grown in this soil in 4-inch varnished pots until they developed eight leaves. Grogan and Zink's (2) method was used, that is the pots were up-ended and the fertilizers applied to the roots in amounts equivalent to 100 pounds of N per acre. The materials used were sodium nitrate, ammonium nitrate, urea, Uramite M, sodium nitrite, and liquid ammoniated ammonium nitrate (the solution often used to supply much of the N in today's mixed fertilizers). Soil analysis made before these applications showed the presence of nitrate-N and ammonium-N in amounts equivalent to 120 and 40 pounds per acre, respectively. Five plants from each treatment were harvested after 2 weeks and the remaining five after 4 weeks. Ammoniated ammonium nitrate and sodium nitrite caused root injury in 2 weeks and the slower-acting urea and Uramite M, which yield ammonia on hydrolysis, caused typical root injury in 4 weeks (Table 1). All check plants were free of injury, as were those treated with ammonium or sodium nitrates.

Table 1. Incidence of injury to lettuce roots grown in steamed muck supplied with nitrogen from different sources.

	Number roots showing indicated degree of injury after application of fertilizers at rate of 100 pounds N/acre							
	After 2 weeks				After 4 weeks			
N source	none	slight	severe	dead	none	slight	severe	dead
$NaNO_3$	4	1			4	2		
Urea	4	1				1	4	
Uramite M	4	1					5	
A ANO_3[a]			3	2			2	3
$NaNO_2$			5				3	2
Check	5					5		
NH_4NO_3	5				4	1		

[a]ammoniated ammonium nitrate, a liquid containing 41% of its N in form of NH_3.

It is believed that varnishing of the pots, which decreased the rate of moisture loss, and the rate of nitrification, was an important factor in creating soil conditions like those in the field.

2. Intolerance of 456 Lettuce in Sand Culture to Ammonium-N:

Plants of the variety 456 were first grown in glazed 1-gallon crocks of quartz sand watered with a complete nutrient solution at pH 5 and containing calcium nitrate as the nitrogen source (7). After 6 weeks the calcium nitrate was omitted and replaced with different amounts of ammoniated ammonium nitrate. All levels of the latter solution above 1000 ppm of N soon killed the plants, 500 ppm induced severe injury, and even at 200 ppm (= 100 pounds ammonium-N per acre) the plants were stunted and roots developed brown corky symptoms and a brown vascular discoloration very typical of symptoms found in the field.

3. Injury to Lettuce Grown Outdoors in 3-Gallon Tin Cans in Flooded and Nonflooded Muck (Steamed and Unsteamed) and Supplied with Different Nitrogen Sources:

A truck load of top muck from an Oswego farm where corky root rot has been a serious problem was brought to Ithaca in July and placed in 300 three-gallon tin cans. One seedling of variety 456 lettuce, sown originally in sterile vermiculite, was planted in each tin can. Some of these tins of muck were autoclaved before planting. Half of the tins were then drenched for 2 weeks under an automatic intermittent mist set up outdoors; presumably, this removed much of the excess soluble nitrogen. The remaining 150 tins received only natural rainfall (.14 inch); of these, 12 had been autoclaved before planting. On September 14, when the plants were fairly mature, nitrogen was applied to groups of 10 plants at two levels, 100 and either 200 or 300 pounds per acre in the form of sodium nitrite, ammonium nitrate, urea, Uramite M, ammoniated ammonium nitrate, or sodium nitrite. Lime at approximately 1000 pounds per acre was added to half the tins in each treatment to see if it would help counteract the injury from NH_4. At this time soil determinations were made of pH, ammonium-N and nitrate-N. No other fertilizer was added and no plants made very good heads. In late October all roots were examined and scored. Data on the effects of the drenching and steaming on ammonium-N, nitrate-N, pH, and root injury are summarized in Table 2 and those on the effect of liming and of the different sources of nitrogen on root rot in Table 3.

Table 2. Soil pH, ammonium-N, and nitrate-N in muck soil (steamed and not steamed) drenched for 2 weeks and not drenched, and relative injury to lettuce roots grown therein.

Treatment of muck	pH	NH_4-N (pounds/acre)	NO_3-N (pounds/acre)	Relative root injury[a]
Drenched				
Steamed	6.0	64	20	2.00
Not steamed	5.4	12	120	.90
Not Drenched				
Steamed	5.3	320	40	5.00
Not steamed	4.9	17	120	.66

[a]Mean score for five plants rated from 0 to 10, where 0 = no rot and 10 = completely rotted off.

According to the data in Table 2, an increase in corky root symptoms might have been related either to an increase in pH, an increase in ammonium-N or a decrease in nitrate-N. The relationship to increased pH can be ruled out because optimum pH for lettuce is 6+. Decreased nitrate-N can also be eliminated. However, as many a grower of vegetables has learned from the use of too fresh manure (from hens, horses or cows), ammonia is well known to be toxic to plant roots. The data also indicated that drenching tended to reduce the concentrations of soluble N, both NH_4-N and NO_3-N, and had a somewhat beneficial effect in reducing corky root symptoms. This is also illustrated by the data for mean scores of corky root (Table 3).

The data in Table 3 show that drenching tended to reduce corky root symptoms but that a grower could not rely on it to eliminate the disease. Liming also tended to reduce severity of symptoms, but not consistently. Of 17 comparisons with and without lime, 12 showed a considerably lower score where lime was applied. The summation of the differences in root scores in favor of lime was over three times as large as the differences in favor of no lime. More consistent results might have been obtained if mixing could have been more thorough than was possible after the plants were set. The highest mean root scores, that is, roots nearly all rotted off, were from plants treated with sodium nitrite, ammoniated ammonium nitrate, and urea, while the lowest were from those treated with Uramite M, sodium nitrate, and ammonium nitrate. The magnitude of the differences between these two groups was

Table 3. Relative severity of corky root symptoms in lettuce roots grown on drenched and nondrenched muck soil supplied with nitrogen from various sources, with and without added lime.

Nitrogen treatment		Relative severity of root symptoms[a]			
	Pounds of	Soil drenched		Soil not drenched	
Source	N/acre	no lime	plus lime	no lime	plus lime
Uramite M	100	3.50	4.50	5.50	3.25
	300	3.75	1.75	4.25	2.25
$NaNO_3$	100	3.75		4.50	
	300	5.33	2.75	5.25	3.75
NH_4NO_3	100	2.50		5.50	
	300	2.75	4.00	5.66	6.00
Urea	100	3.75	4.50	9.00	4.00
	300	7.75	5.50	9.50	8.20
$NaNO_2$	100	9.25	8.00	7.33	7.50
	200	8.75		9.70	
A ANO_3	100	8.33	6.20	9.5	7.66
				10.00	9.12
Mean Scores		5.30	4.65	7.11	5.74
Checks (no N added)		.90	.75	.66	
Autoclaved (no N)		2.00		5.00	

[a]Mean score for five plants rated from 0 to 10, where 0 = no rot and 10 = rotted off.

nearly 100%. Admittedly, the high rating of Uramite M in this outdoor experiment is hard to explain in view of its low rating in the indoor first experiment. However, there was still more corky root rot on plants receiving sodium nitrate than is desirable, which may indicate that much of the toxic potential of this soil had not been removed. Temperatures during the time of this experiment, September-October, were not the most favorable for promoting nitrification. Calcium nitrate would have been desirable in this experiment as one source of nitrogen, in view of Marlatt's work (5) and of the known antagonistic effect of calcium on ammonium absorption.

Fertilizer experiments on mucklands have indicated that applications of nitrogen after June are not really needed for lettuce, since nitrification takes place so rapidly and a crop removes less than 50 pounds of nitrogen per acre.

In general, the results of this experiment support the theory that materials and procedures which increase ammonium-N also increase corky root, whereas those which reduce ammonium have the opposite effect.

4. Ammonium-N and Severity of Corky Root Rot in Several Oswego County Muckland Fields:

By means of a chemical procedure modified for use with muck soil, the senior author analyzed 30 samples of muck for ammonium-N[2]. An interesting relation between corky root on certain farms and the presence of NH_4-N in the fields was found (Table 4). The work thus far has suggested more than 250 pounds of ammonium-N per acre is undesirable for Imperial 456 variety unless the soil reaction can be brought above pH 5.5 (cf. fields 17 to 20).

One of the noteworthy things about these data is the striking difference between the amounts of corky root rot in fields 17 to 20 and those in fields 21 to 24 on the same farm. Both groups were high in ammonium-N, but they differed with respect to pH. Just before planting, fields 17 to 20 received approximately 2 tons of hydrated lime per acre, which brought the soil reaction to about pH 5.6. This suggests that if muck is more acid than pH 5.3, liming before sowing may be helpful, at least on the crop sown during July. Roots from the two portions of this farm are shown in Figure 1A.

[2]The method is based on three important considerations: 1) that the test must be commenced within a half hour of the collection of the field sample, 2) that extraction of the exchangeable NH_4 ion should be done with the sodium ion, and 3) that the objectionable turbidity produced by certain ions insoluble in alkaline solutions but commonly present in muck can be coagulated and thus removed by zinc sulfate and sodium hydroxide. Details of procedure may be had from the authors upon request.

FIGURE 1. A --Upper, roots from field heavily limed before sowing; Lower, not limed.

B -- Longitudinal section through diseased roots showing discoloration and eventual dry rot of vascular region.

C, D, E, F -- Roots from outdoor experiment in 30-gallon tin cans (see Tables 3, 4). C1, two unfertilized checks; C2, same from autoclaved muck; D1, urea 100 pounds per acre; D2, same plus lime; E1, one unfertilized check; E2, sodium nitrate, 100 pounds per acre; E3, same plus lime; F1, ammoniated ammonium nitrate, 100 pounds per acre; F2, same plus lime.

Table 4. Field content of ammonium, pH, and severity of corky root on some Oswego County mucklands.

Field		pH	NH_4-N/acre (in pounds)	Root rot
I	1	5.1	22	Negligible
	2	5.15	26	do.
	3	5.0	24	do.
	4	5.2	26	do.
	5	5.4	44	do.
	6	5.35	43	do.
	7	5.3	45	do.
	8	5.4	42	do.
S	9	5.2	12	do.
	10	5.25	17	do.
	11	5.1	17	do.
	12	5.15	14	do.
	13	5.1	22	do.
	14	5.2	25	do.
	15	5.15	24	do.
	16	5.1	24	do.
J	17	5.6	313	None
	18	5.55	305	do.
	19	5.6	315	do.
	20	5.6	320	do.
	21	5.0	425	Severe
	22	5.05	440	do.
	23	5.1	430	do.
	24	5.05	440	do.
T	25	5.0	463	do.
	26	5.05	465	do.
	27	4.95	475	do.
	28	5.0	470	do.
K	29	5.2	495	do.
	30	5.3	523	do.

DISCUSSION

Since corky root becomes progressively worse on the second and third crops grown in succession, and since all roots are left in the ground at harvest, we first suspected the cause of the disease to be a gradual accumulation of a pathogen. In the light of these experiments and of growers' practices in Oswego County, we now offer a more logical explanation -- accumulations in the root zone of toxic quantities of ammonium. Oswego County is in a snow belt and the mucklands, which are always in low areas where they receive much run off, get heavily packed and thoroughly water-logged during the winter and early spring when nitrification is at its lowest ebb (9). Some Oswego lettuce growers never plow, and none plow in the spring, but merely disc after applying the recommended 1/2 ton of 5-10-15 or half as much 10-10-10 or equivalent. Many apply considerably larger amounts. Forty % of the nitrogen in the fertilizers is in the ammoniated form. Discing mixes it only with the top 3 inches of muck, which is where corky root occurs (6). A second crop sown only 3 months after the first often suffers more than the first if cool, wet weather prevails. The trouble is worst in wet, cool seasons and on shallow acid muck underlain with a tight mineral soil where poor drainage is a chronic condition. The cultural practices mentioned above, cool wet weather, water-logged soil, and low pH all presumably would slow nitrification and favor accumulation of ammonium nitrogen, of which the variety 456 is very intolerant. Apparently the process does not have to proceed for a long time to bring about symptoms. This is all in agreement with the findings of Grogan and Zink (2), who present a good discussion of the plant physio-

logical processes involved in the phenomenon[3].

Experiments are underway with possible control measures, such as cover cropping, liming, and the use of different sources of nitrogen and times of application. The cover crop may have a 3-fold effect: 1) it will compel the farmer to plow in the spring, 2) it will provide an available carbon source for soil organisms and thereby eliminate some of the nitrogen, and 3) the debris will help improve the aeration of the soil and thereby favor nitrification.

If, in making selections for seed production, more attention could be paid to roots and their condition it seems possible that a strain of Imperial 456 more tolerant of ammonium could be developed. Hannon (3) has shown that of some 50 varieties tested in the field the butter head and leafy varieties, such as Big Boston, White Boston, May King, Grand Rapids, Cos, and Simpsons Curled, are much more tolerant than the crisp heading varieties such as Great Lakes, Premier, Iceberg, New York 12 and the popular strains of Imperial like 456 and 615. It is unfortunate that all of those tolerant of corky root have some other weakness, such as premature bolting, susceptibility to brown rib, tip burn, or lack of market acceptability, that may be more difficult to eliminate.

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[3]Thanks are due Dr. Charles Yager of Oswego College of Education for the use of his chemistry laboratory, close to the mucklands, during the summer months.

CONTROL OF POTATO MOSAIC DISEASES BY EXCLUSION

James W. Guthrie[1]

Plant diseases can usually be controlled by one or more of the following procedures: exclusion, eradication, protection, and host resistance. In 1942 the application of one of the principles of exclusion -- that of voluntary inspection and certification -- was instituted by the Idaho Seed Potato Association in an effort to improve the quality of potato seed. It was soon found that even in such a program the normal procedures of inspection may be inadequate because symptoms of several virus diseases are masked when unfavorable weather conditions prevail, thereby limiting detection of affected plants.

In Idaho, because of the prevalence of intense sunlight throughout the summer season, crinkle mosaic (Potato virus X and A) becomes somewhat masked and thus ready detection of the disease is prevented (Fig. 1). The same conditions do not apply to the potato leafroll virus

FIGURE 1. Symptom expression of crinkle mosaic grown during the summer at Aberdeen, Idaho.

FIGURE 2. Symptom expression of crinkle mosaic grown during the winter at Oceanside, California.

[1]Assistant Plant Pathologist, University of Idaho, Branch Experiment Station, Aberdeen, Idaho. Published with the approval of the Director as Research Paper No. 485 of the University of Idaho, Agricultural Experiment Station, Moscow, Idaho.

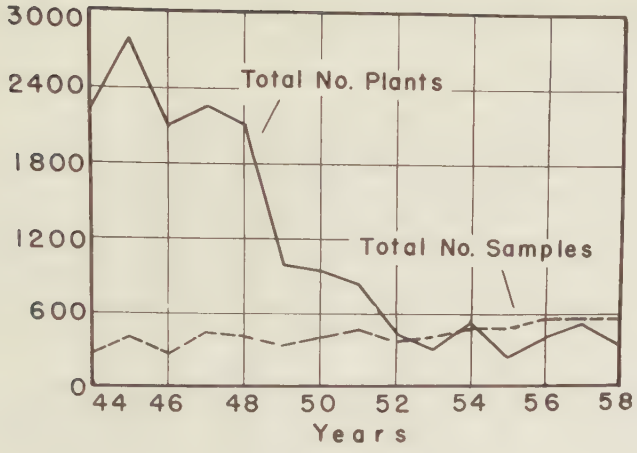

FIGURE 3. The number of potato plants exhibiting mosaic symptoms and the number of samples in southern California winter tests from 1944 to 1958.

disease, however, since prolonged sunlight appears to intensify rather than to mask its symptoms.

Each year since 1943, the Idaho Crop Improvement Association, Idaho's potato seed certification agency, has acquired a 300-tuber sample from each seed grower who intended to raise a crop for certification the succeeding year. When the samples were collected tubers were treated with ethylene chlorohydrin to promote sprouting, shipped to Oceanside, California, and planted during December. The following March a representative of the Idaho Crop Improvement Association inspected the resulting plants. If at the time of this inspection any sample showed more than 2% of the plants infected with any mosaic virus, the parent seed stock was rejected for replanting.

Environmental conditions, such as low light intensity, found in southern California from January to March seemed to be conducive to the expression of potato mosaic disease symptoms (Fig. 2). The data in Figure 3 show that 265 tuber samples were planted in 1944 which produced 2233 plants that exhibited mosaic disease symptoms. During the next 15 years, as a result of the ease of detection and elimination of seed stocks with excessive disease, the total number of mosaic-diseased potato plants in the California plots decreased from 2233 to 346, although the total number of samples tested increased from 265 to 549. This means that in 1944 there was an average of eight mosaic plants per sample whereas in 1958 this number was reduced to 0.1 per sample. This, then, represents an outstanding example of plant disease control by exclusion.

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RESISTANCE IN SWEETPOTATO TO THE INTERNAL CORK VIRUS

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Summary

The disease reaction of 43 sweetpotato clones to the internal cork virus was investigated. Bedding roots of the clones were core-graft inoculated and sprouts from the inoculated roots used to produce roots for evaluating disease reaction. Thirty-two clones developed internal root necrosis; however, the clones differed in the severity of necrosis in their roots. Nearly all roots of the very susceptible clones developed internal necrosis, while few or no roots of others developed the symptom. This extreme range in the development of the necrotic symptom in roots of the several clones suggests the presence of genetic factors in sweetpotato affecting internal necrosis.

No internal necrosis was produced in the roots of 11 clones. When root tissue from the symptomless clones was core-graft inoculated to virus-free Porto Rico roots, the internal cork virus was demonstrated in seven (symptomless carriers), doubtful in three, and absent in one. This response of the symptomless clones suggests that genetic factors of the sweetpotato also influence virus multiplication within plant tissue.

The inheritance of the necrotic and virus multiplication expressions of resistance to the internal cork virus was studied in progeny from a cross, susceptible X resistant, and in progeny from the resistant parent selfed. The progeny from the cross (60 clones) were distributed as follows: 40, susceptible; 17, symptomless carriers; and 3, resistant to virus multiplication. The distribution of progeny from the resistant parent selfed (36 clones) was 5, susceptible; 10, symptomless carriers; and 21, resistant to virus multiplication. These data indicate that resistance to the internal cork virus should not be difficult to obtain in a sweetpotato breeding program.

Early in the history of the internal cork virus disease of sweetpotatoes it was observed that roots of some sweetpotatoes infected with the virus did not develop the characteristic internal necrosis (10, 11). These observations were confirmed by core-graft inoculating bedding roots of clones with virus-infected tissue and examining roots produced by the new crop of sprouts (2, 4, 6, 8). It was also demonstrated that symptomless clones contain the virus by cleft and core grafting stem and root tissue from infected symptomless clones to internal-cork-free Porto Rico (1, 2, 8). The objectives of the investigation herein reported were to evaluate existing varieties and breeding clones for their resistance to the internal cork virus, and determine if the resistance could be transmitted to progeny. A brief report on the first objective was published earlier (9).

MATERIALS AND METHODS

Forty-five sweetpotato clones were used for evaluating resistance. These included sweetpotato varieties grown commercially in North Carolina, clones in the Regional Trials of the Sweetpotato Cooperators Group, and breeding clones from various investigators[1]. Two clones were duplicated from different investigators, thus only 43 clones were studied.

Roots of all clones were inoculated by core grafting into them infected root tissue from Porto Rico potatoes. The core-graft inoculations were made prior to bedding for sprout production each spring. Sprouts from core-graft inoculated roots were planted in alternate rows with infected Porto Rico plants to provide inoculum for natural spread of the virus in case sprouts from inoculated roots failed to become infected with the virus. Harvested roots were incubated at temperatures between 75° and 85° F for 5 to 7 months before they were examined for internal necrotic symptoms.

Those clones that failed to produce necrotic lesions in their roots and many that produced

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[1]The following investigators contributed clones for this study. The abbreviations used to identify these sweetpotatoes in subsequent tables are included in parentheses: Dr. M. B. Hughes, Blackville, South Carolina (SC(B)); Dr. O. B. Garrison, Clemson, South Carolina (SC(C)); Dr. Teme P. Hernandez, Louisiana (La); Dr. E. E. Steinbauer, Maryland (USDA); Mr. Marshall Deonier, Mississippi (USDA(M)). Clones originating in North Carolina (NC) and Oklahoma (Okla) were also evaluated.

few necrotic lesions were further studied by core grafting tissue from their roots into roots of internal-cork-free Porto Rico. In this case the inoculated Porto Rico roots were incubated for 5 to 7 months at 75° F and examined for internal cork symptoms (8).

The internal necrotic root symptom was used for disease diagnosis.

RESULTS

The sweetpotatoes were first categorized into two groups -- those with internal root necrosis and those without internal root necrosis. Thirty-two clones produced necrosis in their roots. These sweetpotatoes, with parentage when known, are arranged in Table 1 from the most susceptible to least susceptible as indicated by the percentage of their roots having necrotic symptoms. The percentage values are relative as some of the more susceptible clones were tested only 1 year, and other clones produced fewer roots or the roots deteriorated more extensively during warm temperature storage. The sweetpotatoes differed widely in their development of the necrotic symptom. Nearly all roots of the susceptible variety Porto Rico developed the necrotic symptom. From this extreme of susceptibility, the percentage of roots with symptoms in the different clones diminished to practically zero. Clones having fewer than 5% of their roots with symptoms would probably develop few, if any, internal lesions when stored at recommended temperatures (55° to 60° F) and might be considered, on a practical basis, as symptomless carriers.

The disease reaction of a number of clones in Table 1 has been reported by other investigators, and there is a general agreement with the data reported here. Clones E-7, 11-20, and B5999 were classed as symptomless carriers (4). This report is based upon 1 year's data and a 90-day warm-temperature storage. Goldrush, Heartogold, and Earlyport were reported as susceptible while Allgold was resistant (2). Although Earlyport is classified as susceptible, a smaller percentage of its roots develops internal necrosis than do the roots of Porto Rico (6). The percentage of Earlyport roots with lesions in Table 1 is smaller than the value given earlier (6).

The wide range of symptom development in roots of different clones indicates the presence of genetic factors that affect the development of the internal necrosis symptom. Some clones appear to have this resistance to a high degree, as few or no necrotic symptoms developed in their roots.

Internal root necrosis was not found in roots of 11 clones. Tissue from roots of these clones was core grafted to internal-cork-free Porto Rico roots, and following incubation the Porto Rico roots were examined for the presence of necrosis. In addition, all clones in Table 1 below Triumph were similarly core grafted to Porto Rico to confirm the diagnosis of the presence of cork. The results are tabulated in Table 2.

Internal root necrosis developed in many Porto Rico roots inoculated with root tissue from the different clones. Root tissue from all clones that exhibited root symptoms (Table 1) also induced cork lesions in core-graft inoculated roots of Porto Rico. Only six of these symptom-producing clones are included in Table 2 to compare with data obtained from the symptomless clones. The symptomless clones are arranged in a descending order of the percentage of core-graft inoculated Porto Rico roots that developed symptoms. All Porto Rico roots inoculated with tissue from Nancy Hall roots developed cork lesions. The percentage of Porto Rico roots developing internal necrosis ranged from this high incidence to zero for roots inoculated with tissue from roots of H. M. -2. Lesions that developed in the Porto Rico roots inoculated with tissue from N. C. 164, H. M. -36, and H. M. -15 were few in number and small and the diagnosis was doubtful. Clone H. M. -2 was tested 4 years, and the absence of lesions in Porto Rico roots indicates that the virus, if present, was at a low concentration in root tissue of this clone.

This range in percentage of Porto Rico roots that developed internal necrosis following core-graft inoculations with tissue from roots of symptomless clones is similar to that noted in Table 1. This response is interpreted as reflecting the virus concentration in the root tissue of the several clones. Differences in virus concentration possibly reflects genetic factors that inhibit virus multiplication. Nancy Hall is susceptible to virus multiplication while H. M. -2 is resistant to virus multiplication.

These data indicate two expressions of resistance in sweetpotatoes to the internal cork virus: resistance to development of necrosis, and resistance to virus multiplication. Both resistances appear to be expressed quantitatively.

The pedigrees of the clones reveal some interesting relationships. Nancy Hall, which is resistant to necrosis, is a parent of several clones resistant to necrosis or virus multiplication. The variety Pelican Processor is also reported resistant to necrosis (2) and it is a parent of

Table 1. Sweetpotato clones, parentage, and the percentage of their roots with internal cork symptoms after storage at 70° to 80° F.

Clone	Parentage	Source	Years tested	Roots examined	% roots with symptoms
Porto Rico	Derived from Mameyita	NC	4	566	93
262-27	(98 x Yellow Strasburg) x (Yellow Yam O.P. x Nancy Hall)	SC(B)	1	54	85
150	Mutant of L-69 (Porto Rico x 47742)	SC(C)	1	101	70
Goldrush	(Mameyita x L-4-6) x (Pelican Processor x Triumph)	La	3	299	57
Heartogold	Mameyita x Yellow Yam	La	1	100	47
Triumph	Old variety	NC	1	120	38
B6091	Porto Rico x (Pelican Processor x Triumph)	USDA	4	97	34
B5941	(Yellow Yam x Nancy Hall) x (Processor x Triumph)	USDA	5	209	33
47-60	(U.S. R. 135762 O.P.) from Cuba, O.P.	SC(B)	4	299	29
H.M.-18	(Yellow Yam x Nancy Hall) x (Porto Rico x Pelican Processor)	USDA(M)	2	94	25
L-21	Mameyita x L-4-6	La	4	263	24
Australian Canner	Australian Sel. from Hawaiian seed		4	116	19
Earlyport	(Mameyita x L-4-6) x (Pelican Processor x Triumph) - 45-2	La	2	168	14
276	Porto Rico x Clemson 26 (unknown)	SC(C)	4	198	12
11-20	(98 x Yellow Strasburg) x (Yellow Yam O.P. x Nancy Hall)	SC(B)	4	84	10
B6018	(Mameyita x L-4-6) x ((Porto Rico x Triumph) x Mameyita))	USDA	4	377	8
White Star	Hawaiian variety Laupahoehoe-O.P.	USDA	3	127	8
Hayman yam	Old variety	NC	4	247	6
Orlis	Mutant of Little Stem Jersey		4	211	5
Oklahoma 2	Porto Rubio (mutant of Porto Rico) - selfed	Okla.	3	44	5
B6016	Same as B6018	USDA	4	346	3
Allgold	(Creole selfed x Triumph - O.P.) O.P.	Okla.	4	318	3
B4570	Nancy Hall x Porto Rico	USDA	4	251	3
L-155	Pelican Processor x Triumph	La	4	199	3
P.I. 153655	From Tinian Island		3	89	2
P.I. 153909	Japanese variety Taihaku Saitama No. 1		4	110	2
H.M.-38	(Porto Rubio, selfed) O.P. x Australian Canner	USDA(M)	4	409	1
E-7	(Yellow Yam O.P. x Nancy Hall) x (98 x Yellow Strasburg)	SC(B)	4	96	1
L-244	Porto Rico x Pelican Processor	La	4	195	1
B5999	(Yellow Yam x Nancy Hall) x (Pelican Processor x Triumph)	USDA	4	119	1
N.C. 41 x 20-3	((Yellow Yam x Nancy Hall) x 6-42-1) x unknown	NC	4	136	1
N.C. 41 x 20-18	((Yellow Yam x Nancy Hall) x 6-42-1) x unknown	NC	4	319	1

Table 2. Incidence of internal cork symptoms in virus-free Porto Rico roots core-grafted with tissue from roots of sweetpotato clones.

Clone	Parentage	Source	Backgraft to Porto Rico: Roots with symptoms[a]	%
Clones having root symptoms (Table 1):				
Porto Rico			15/16	94
Hayman yam			5/11	46
Allgold			8/12	75
L-244			9/11	82
B5999			4/7	57
NC 41 x 20-18			3/12	25
Clones without root symptoms:				
Nancy Hall	Old variety	NC	10/10	100
N.C. 41 x 20-9	(Yellow yam x Nancy Hall) x 6-42-1) x ?	NC	6/9	67
259	Unknown	SC(C)	8/14	57
Norton yam	Old variety	NC	6/12	50
Okla. 1	Creole selfed	Okla	5/10	50
11-136	(98 x Yellow Strasburg) x (Yellow Yam O.P. x Nancy Hall)	SC(B)	5/12	42
B6077	(Pelican Processor x Triumph) x (Yellow Yam x Nancy Hall)	USDA	2/15	13
N.C. 164[b]	Unknown	NC	2/23(?)[c]	9 (?)
H.M.-36	(Porto Rubio-selfed) O.P. x PI 153655	USDA(M)	1/14(?)	7 (?)
H.M.-15	(Porto Rubio-selfed) O.P.	USDA(M)	1/17(?)	6 (?)
H.M.-2	(Creole x Porto Rico) x Porto Blanco	USDA(M)	0/21	0

[a]Numerator, Porto Rico roots with lesions; denominator, inoculated Porto Rico roots examined.
[b]Erroneously designated L-240 in an earlier paper (8).
[c](?) lesions few in number and less than 1 mm in diameter. Accuracy of diagnosis was doubtful.

resistant clones in both categories. Oklahoma 2, a clone exhibiting considerable resistance to necrosis, was selected from a selfed population of Porto Rubio. The latter variety is a somatic mutant from highly susceptible Porto Rico (7). This suggests that Porto Rico either contains some factors for resistance to necrosis, or that the somatic mutation also resulted in improved resistance to necrosis.

Sixty clones from the cross Porto Rico (susceptible) x H.M.-15 (resistant to virus multiplication) were selected for horticultural characters and then evaluated for internal cork resistance. Progeny from H.M.-15 selfed were also evaluated for resistance. Many of the 66 clones from H.M.-15 selfed were poor root producers and 30 clones were lost in warm temperature incubation. The progeny from both family lines were studied for 2 to 4 years by the procedures described earlier, and disease reactions of clones from both lines are summarized in Table 3.

Table 3. Internal cork reaction of progeny from a cross resistant x susceptible and the resistant parent selfed[a].

Parentage	Clones tested	Susceptible	Resistant to: necrosis	Resistant to: virus
H.M. 15 x Porto Rico (Res.) x (suscept.)	60[b]	40	17	3
H.M. 15 selfed (resistant)	36	5	10	21

[a]The resistant parent H.M. 15 was the only clone that bloomed in the field and seed were collected.
[b]These clones kindly provided by Marshall Deonier, Meridian, Mississippi.

The following criteria were used in classifying the disease reaction of the clones. Those clones having more than approximately 5% of their roots with cork symptoms were classified susceptible. Clones having approximately 5% or less of the roots with necrotic lesions and the virus was demonstrable in the root by core grafting tissue to Porto Rico roots were classified as resistant to necrosis. Clones having no necrosis and the virus was not demonstrable in approximately 10% or less of the core grafted Porto Rico roots were classified as resistant to virus multiplication.

Clones from the cross Porto Rico x H. M. -15 were predominantly susceptible to necrosis. Nearly two-thirds of the clones were in this category while only 5% (three clones) were resistant to virus multiplication. The distribution of progeny from H. M. -15 selfed is practically the reverse. Nearly 60% of the clones were resistant to virus multiplication; whereas, only five, or 14% were susceptible to necrosis, with the remainder resistant to necrosis.

DISCUSSION

The use of internal-cork-free Porto Rico roots as an indicator for the presence of internal cork virus in symptomless potatoes has a number of limitations. The procedure is slow and laborious. Small lesions, a millimeter or less in diameter, are difficult to diagnose. The incubation period of 5 to 7 months at 75° F for core-graft inoculated Porto Rico roots may be inadequate to obtain an expression of necrosis if the virus content of the inoculum is low. A longer incubation period may give a different reading for some of the clones classified as resistant to virus multiplication. A more sensitive and rapid procedure for diagnosing the presence of virus in clones is needed. The Scarlet O'Hara morning glory (3) proved ineffective in this work as an indicator of the internal cork virus.

The survey of the sweetpotato clones provides several categories into which they may be separated according to their reaction to the internal cork virus. First, there are clones which produce necrotic lesions when the virus is present and may be classified as susceptible types. Second, certain clones contain the virus, as indicated by core grafting to virus-free Porto Rico roots, but do not produce the necrosis; these may be classified as the symptomless-carrier types. Third, there are those which upon inoculation with the virus produce no necrotic lesions and upon core grafting to virus-free Porto Rico roots show little or no transfer of the virus; these are the resistant types. The concentration of virus in the clones tested, the compatibility of cores from different clones with virus-free Porto Rico roots and the equal transfer of virus from cores into virus-free roots are factors that may affect the expression of the internal cork virus and influence the above categories.

Inheritance studies and genetic patterns in the sweetpotato, a vegetatively propagated plant, are complicated as the plant is a hexaploid (5) and is normally highly heterozygous as evidenced by various segregations encountered. It has been postulated (12) that the sweetpotato originated as an allopolyploid. Secondary association of some bivalents suggests that total differentiation of the sweetpotato chromosomes has not reached the stage where the chromosomes behave as in diploids, the condition found in many natural polyploids. Genetic factors affecting the two expressions of resistance to the internal cork virus, necrosis and virus multiplication, may be associated in their inheritance or they may be inherited independently. Necrosis is the ultimate expression of the virus in affected root tissue and, a priori, this symptom will develop only in clones that are susceptible to virus multiplication. If the sweetpotato behaves as a diploid, one genetic factor may affect necrosis and another virus multiplication. The interaction of these factors may result in the intermediate expression -- symptomless carriers. An equally acceptable explanation of the resistance expressed is a single locus diallelic system wherein resistance is due to dosage effects of alleles of the same chromosome. In progressing from resistance to susceptibility, a number of one type of allele may result in susceptibility to virus multiplication and more of the same allele susceptibility to necrosis.

The disease reaction of the progeny from the two family lines (Table 3) indicates some interesting aspects of resistance as well as what might be expected in a breeding program of crossing a resistant with a susceptible parent. The proportion of progeny from the cross susceptible x resistant that were resistant to necrosis (symptomless carriers) suggests that this resistance will not be difficult to obtain in a breeding program. Resistance to virus multiplication from such a cross can be obtained but will require larger populations. It is obvious that H. M. -15 is not homozygous for resistance to virus multiplication or necrosis. Assuming the resistance factors are not recessive, a cross employing two clones resistant to virus multiplication would probably yield a high proportion of clones with this resistance.

From the standpoint of introducing an internal cork resistant variety, the most desirable

clone to release would be one having the high level of resistance to virus multiplication. A clone resistant to necrosis but susceptible to virus multiplication, if infected and grown in close proximity to virus-free susceptible sweetpotatoes, would provide a reservoir of inoculum for insect vectors to inoculate the susceptible potatoes. This would necessitate propagating virus-free susceptible varieties and infected symptomless carrier types separately in order to maintain seedstock of the susceptible varieties free from internal cork.

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CERATOCYSTIS COERULESCENS ON SUGAR MAPLE IN THE LAKE STATES

K. J. Kessler, Jr. and R. L. Anderson[1]

The fungus Ceratocystis coerulescens (Münch) Bakshi, not previously reported on hardwoods in the Lake States region, was found in 1959 on a sugar maple tree in the Upper Peninsula Experimental Forest at Dukes, Michigan. Early in the spring the tree, measuring 26 inches in diameter at breast height and about 95 feet tall, was observed to have abnormally small leaves[2]. During the growing season leaf development was observed periodically. The leaves never attained more than one-half normal size but remained green all summer.

The condition of the tree was called to the attention of the authors during an early winter visit to the area. Examination of buttress roots revealed a dark greyish-brown, longitudinal streaking of the wood, with occasional green and red streaking also present, particularly at the extremities of the streaked areas (Fig. 1). Similar streaking was also present at breast height in the trunk (Fig. 2).

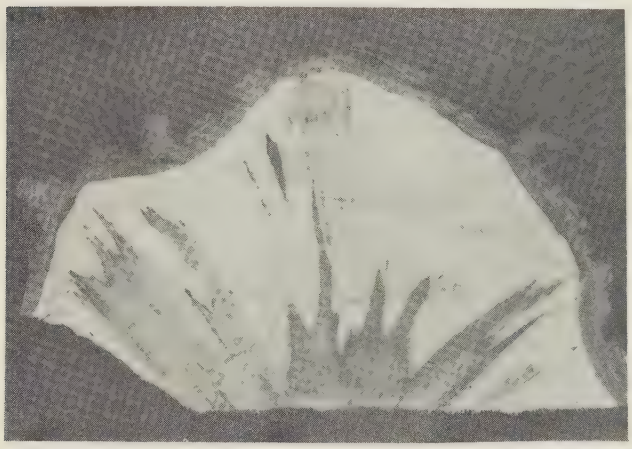

FIGURE 1. Cross section showing streaking of the wood cut from a buttress root. The section has been moistened with water to show the detail of the streaking more clearly.

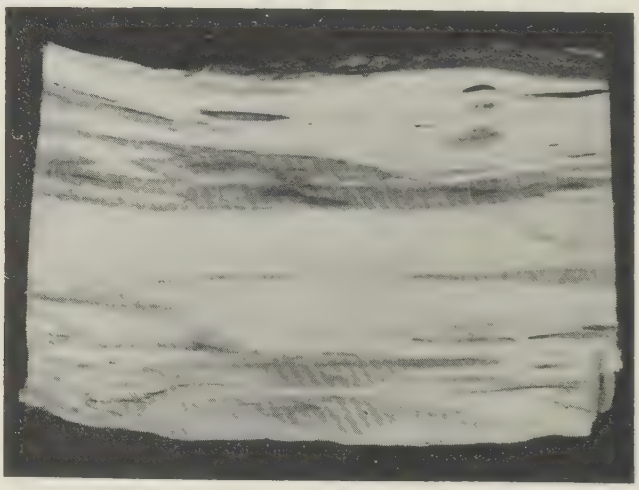

FIGURE 2. Longitudinal section showing streaking of the wood at breast height. The section has been moistened with water to show the detail of the streaking more clearly.

Isolations were made from streaked wood of buttress roots and trunk by dipping small segments of the wood into 95% ethyl alcohol, flaming them, and then placing chips cut from the segments onto malt agar plates. These isolations resulted in the recovery of Ceratocystis coerulescens (Münch) Bakshi, the causal agent of the sapstreak disease of maple in the

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Southeast (3). The fungus was also isolated from streaked wood by placing the wood in a moist chamber so that perithecia would develop on the surface of the wood. Ascospores picked from the tips of the perithecial necks and plated on agar produced cultures similar to the ones isolated from the wood. Perithecia never developed on wood taken from the same tree from nonstreaked areas and placed in a moist chamber.

Morphological and cultural characteristics of the fungus agree with those described by Davidson (2), Hepting (3), and Hunt (4). Characters which readily identify the fungus as being the form of C. coerulescens found on hardwoods are: 1) The distinctive, long, dark, slender, unbranched hyphae arising from the bases of the perithecia (Fig. 3); 2) The two general kinds of endoconidiophores present -- one long and tapered (Fig. 4), the other shorter and untapered; 3) The typical sweet, musty smell of the fungus when grown on malt agar; and 4) The rapid growth of the fungus on agar plates.

FIGURE 3. Perithecium. Note mass of ascospores at the tip and hyphal appendages arising from the base of the perithecium.

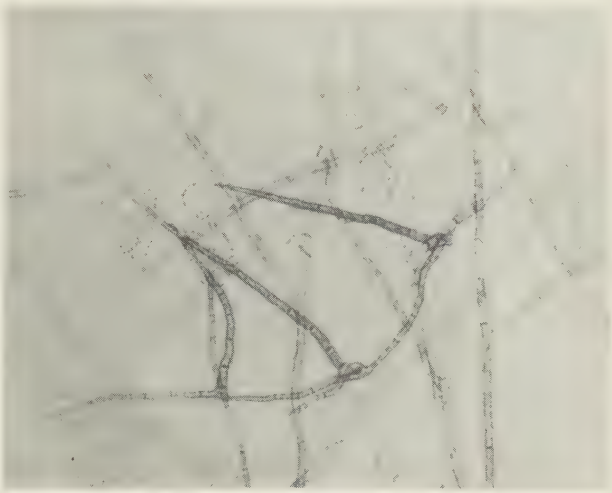

FIGURE 4. Three endoconidiophores from which endoconidia are being extruded.

DISCUSSION

Hunt (4), in his monograph on the genus Ceratocystis, regarded C. coerulescens on hardwoods (which had been previously described by Davidson (2) as Endoconidiophora virescens) as being synonymous with the original C. coerulescens from conifers. The basis for placing these two fungi in synonymy was the lack of morphological difference between the two. Cultures of the form on conifers, however, have the odor of banana oil while those from hardwoods do not. Birkinshaw and Morgan (1) have investigated the volatile metabolic products that could cause the banana oil odor of the coniferous form and have found that isobutyl acetate was the material responsible. They were unable to detect isobutyl acetate in cultures of the hardwood type. Birkinshaw and Morgan's work thus provides a chemical basis for separating the form isolated from conifers and the one from hardwoods.

Unfortunately, there have been no cross-inoculation studies to determine if the host ranges of the two forms overlap to any extent. From the work reported here and that of others, it would seem desirable to keep the C. coerulescens type on hardwoods separated from the one on conifers as a distinct form of C. coerulescens.

The severe localized mortality caused by maple blight in northeastern Wisconsin (5), plus fairly extensive maple dieback, is responsible for considerable concern in the Lake States region regarding the possible role of maple pathogens in these problems. C. coerul-

escens has been reported to be pathogenic in the Southeast (3). Although other investigators have made an intensive search for possible pathogens causing maple blight, this is the first report of the hardwood form of C. coerulescens in the Lake States. The probabilities are very remote, therefore, that it is involved in the maple blight problem. Furthermore, discovery of the fungus in a single tree does not provide any evidence that it is responsible for the rather extensive and severe maple dieback now occurring in the region.

The discovery does prove that a fungus is present in the region that is known to be capable of being pathogenic on maple. Present plans are to make a thorough investigation of its possible role in the dieback problem.

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LAKE STATES FOREST EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

OAK DIEBACK IN VIRGINIA IN 1959

John S. Boyce, Jr. and Charles F. Speers[1]

In September 1959 the Southeastern Forest Experiment Station received reports of extensive branch dieback in chestnut oak (Quercus prinus) and white oak (Q. alba) in western Virginia. Examination of dead and dying twigwood from Craig and Shenandoah counties showed that pycnidia of a Dothiorella were consistently present. Lesions caused by the pit-making oak scale Asterolecanium sp. (probably variolosum) occurred on many affected twigs, and on some of them Dothiorella pycnidia were present only within bark areas killed by the scale. This suggested that in these cases the fungus invaded the dying or dead bark following scale attack. However, it appeared that fungus infection was independent of scale injury on other twigs.

Single conidial cultures of the Dothiorella were obtained. Dr. J. C. Carter identified the fungus in culture and the twigwood pycnidia as Dothiorella quercina (Cke. & Ell.) Sacc. He has shown this fungus to be commonly associated with branch cankers and dieback in both red and white oaks in Illinois, and its pathogenicity was established by wound inoculation tests[2].

Heavy infestations of the pit-making oak scale are capable of killing twigs, branches, and entire trees, especially on poor sites or when infestations occur during periods of drought[3]. However, only light-to-medium scale populations were observed on the Virginia material.

Some mortality of affected chestnut oak and white oak was seen October 7, 1959 along a ridge at Edinburg Gap in Shenandoah County. The fungus-scale infestation was common here, and it is possible that its effect was aggravated by a drought in June. No unusual mortality of the same oak species was noted in a stand near Jerome in the same county, where the oaks are growing on gently sloping land at a lower elevation. The fungus-scale infestation was prevalent and had caused branch dieback and crown thinning in this stand.

The relative importance of the fungus and the scale in causing this oak dieback is not clear, since both organisms can kill twigs when acting alone. It is likely that their close association increased the amount of damage. Scale infestations may have permitted more abundant Dothiorella infections by creating suitable bark lesions for entrance by the fungus.

Possibly D. quercina, the scale, or both have been contributory causes to the unexplained decline of white and chestnut oaks in parts of western Virginia in recent years.

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TERRACLOR CONTROLS OLPIDIUM ON LETTUCE

Saul Rich

Abstract

Terraclor 75% mixed dry with sand at 50 ppm completely protects lettuce seedlings against infection by Olpidium. Zinc at 50 ppm and 200 ppm does not. Terraclor, already cleared for use on lettuce, may prove to be immediately useful for the control of big-vein reported by Grogan et al. (1) to be associated with Olpidium infection of lettuce roots.

Grogan et al. (1) have found a strong association between big-vein of lettuce and infection of lettuce roots by Olpidium brassicae. Treatments to protect lettuce against the soil-borne Olpidium may prevent big-vein. The effectiveness of Terraclor (75% pentachloronitrobenzene) against another chytrid, Plasmodiophora brassicae, suggested its possible action against Olpidium. Zinc sulfate was also tried because Rich (3) had earlier reported it as a chemotherapeutant for big-vein. In addition, Tomlinson (5) used zinc to control crook root of watercress caused by still another chytrid, Spongospora. Rich (4) also reported that zinc had some effect against club root.

METHOD

The Terraclor and zinc sulfate were mixed dry with dry sand. Each treatment consisted of five pots. Terraclor was used at 50 ppm. $ZnSO_4 \cdot 7H_2O$ was used at 50 and 200 ppm of actual zinc. Each pot received 25 cc of soluble fertilizer, after which two seedlings of lettuce, variety Great Lake, were planted per pot. The following day, July 14, each pot was inoculated with 25 ml of inoculum made from roots of field-grown lettuce plants showing big-vein symptoms. To make the inoculum, the roots were ground in a Waring Blendor and made up with distilled water to give an amount sufficient for the experiment. The roots of the seedlings were examined for Olpidium on August 25.

RESULTS

Probably because of the warm greenhouse temperatures, no big-vein symptoms developed in any treatments. The Terraclor treatment, however, completely protected the roots from Olpidium infection. None of the 10 plants grown in the Terraclor treated sand showed any Olpidium. In both zinc treatments and in the untreated check 8 out of 10 plants showed heavy infection with Olpidium. Only the plants grown in 200 ppm of zinc showed any injury. These plants were appreciably stunted.

DISCUSSION

Terraclor can completely protect lettuce from Olpidium infection, and if Olpidium infection is indeed necessary for the production of big-vein symptoms, Terraclor should protect lettuce against big-vein. This treatment would prove useful immediately, because Terraclor is cleared for use on lettuce to control other diseases such as bottom rot and drop rot. Experiments are now in progress to determine if Terraclor will prevent big-vein.

Zinc mixed with sand at the time of planting lettuce does not protect against Olpidium infection. Marlatt (2) has reported that applying extra zinc to lettuce plants in the field does not prevent big-vein. It may be, then, that in order to be effective zinc must be applied to lettuce plants for a period prior to their being set out in infected soil.

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THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN

INFECTIVITY DIFFERENCES BETWEEN OLPIDIUM FROM ROOTS OF SPINACH AND LETTUCE

Saul Rich

Roots of lettuce and spinach, both infected by Olpidium, were collected from a single farm. The lettuce plants showed big-vein symptoms. The specimens were taken from adjacent fields which in previous years had been cropped to both lettuce and spinach.

All stages of Olpidium were present in the roots of both crops, and the fungus in lettuce appeared morphologically indistinguishable from the fungus in the spinach.

Cross inoculations were made using seedlings of lettuce, variety Great Lake, and of spinach, variety Bloomsdale Long Standing. The seedlings were grown in sand. The inoculum was prepared by macerating the washed root tissue in a Waring Blendor, and adding distilled water to make up adequate amounts for the experiment. Twenty-five ml of inoculum were added to each pot. Each treatment was applied to seven pots, with one plant per pot. The plants were inoculated on December 18 and all data taken on February 20.

Of the seven lettuce plants inoculated with lettuce-root inoculum, five had Olpidium infection and the same five plants showed big-vein. Only one of the seven lettuce plants inoculuated with spinach root tissue was infected with Olpidium, and none of these plants showed big-vein symptoms.

All of the seven spinach plants inoculated with spinach root tissue became infected with Olpidium. None of the seven spinach plants inoculated with lettuce root tissue became infected with Olpidium. The foliage of all the spinach plants appeared normal.

Because of the association of Olpidium with big-vein[1], it is important to know if there are strain or species differences. Either the fungi reported here are separate species of Olpidium or physiological strains of the same species.

THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN

[1]Grogan, R. G., F. W. Zink, Wm. B. Hewitt, and K. A. Kimble. 1958. The association of Olpidium with big-vein disease of lettuce. Phytopathology 48: 292-297.

THERMOTHERAPY FOR ROOT-KNOT NEMATODES, MELOIDOGYNE SPP., OF SWEETPOTATO AND TARRAGON PROPAGATING STOCKS

Ivan J. Thomason, S. D. Van Gundy, and H. E. McKinney[1]

Abstract

Dry heat treatment of sweetpotato seed roots at 113° F for 30 hours resulted in complete control of root-knot nematodes in the roots. Sixty-eight % of the roots were still viable and capable of producing "slips" after heating. Hot water treatment (113° for 25 minutes) of tarragon (Artemisia dracunculus) rhizomes infected with root-knot nematodes (Meloidogyne hapla) gave only partial control of the nematodes and resulted in a 32% reduction in viability of the rhizomes.

SWEETPOTATOES

Root-knot nematodes, Meloidogyne incognita and M. incognita acrita, are serious pests of sweetpotato, Ipomoea batatas, in California. Sources of root-knot nematode infestation in the commercial crop are infested propagating beds, seed roots, and/or field soils. Nematodes in the field soils and soil used in propagating beds can be controlled with nematocides. Obtaining nematode-free seed roots has been a problem confronting sweetpotato growers.

The crop is normally propagated vegetatively from seed roots stored through the winter and commonly saved from a portion of the grower's own crop or obtained from other commercial growers. Much of this seed contains viable root-knot nematode eggs. Three out of six lots of sweetpotato seed obtained from growers in San Bernardino County in 1958 contained viable root-knot nematodes. Sweetpotato seed can be freed of root-knot nematode by propagating vine cuttings in clean soil or by hot water treatment of the seed roots prior to bedding. However, the use of vine cuttings or hot water treatments requires considerable effort and expense on the part of commercial growers in order to obtain nematode-free stock.

Burke and Tennyson (1) working in Oklahoma developed the hot water treatment for sweetpotato roots. They suggested treating the seed in hot water at 116° F for 65 mintues. However, Dr. F. Ben Strubble (personal communication) indicated that this treatment has not been widely practiced in Oklahoma.

In 1916 Frandsen (2) reported that a root-knot nematode in potatoes, Solanum tuberosum, was destroyed by placing the infected tubers in an incubator at 40° C for 23 hours. The tubers were still viable after this treatment.

The results of the above-mentioned test suggested that the curing room commonly used by growers to cure and store their seed roots might be utilized to eliminate root-knot nematode from the roots. The curing rooms are equipped with heaters to raise the room temperature to 85° to 90° F for several days immediately after harvest to accelerate suberization of roots injured during harvest. Tests were initiated to determine what temperatures and exposure time would be required to kill the nematodes in infected roots and what effect heating would have on the ability of roots to produce "slips."

MATERIALS AND METHODS

Forty sweetpotato roots showing severe nematode cracking were selected for the first test. The roots were divided into four lots of ten roots each. A section approximately 1/4 the length of each of five roots in each lot was cut off and macerated in a Waring Blendor. The ground root was then mixed with clean soil and planted to a tomato seedling. The remaining 3/4 of the cut roots and the five whole roots were then dry-heat treated in four small dry type, gravity convection incubating ovens. The four lots were treated at oven temperatures of 100°, 105°, 110° and 115° F for 41 hours. Temperature of the roots was determined by placing the bulb of a thermometer in the center of one root in each oven. After treatment another 1/4 section of the cut roots was ground and mixed with soil and planted to a tomato seedling. The whole roots were bedded in sand and maintained at 85° to determine the influence of treatment on viability

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The assistance of Mr. D. C. Purnell, Farm Advisor, University of California Agricultural Extension Service, San Bernardino County, is gratefully acknowledged.

of the roots. After 6 weeks tomato seedlings were removed and the amount of galling on the roots was used as a measure of the viability of root-knot nematodes in the sweetpotato roots before and after treatment. The "slips" from the whole roots were counted after 4 weeks and checked for nematode galls.

The procedure in subsequent tests was similar except that in the last two tests reported a large electrically heated drying oven was used to heat the roots. The oven was equipped with a thermostat for temperature control and fans for circulating the air within the oven. The sweetpotato variety Velvet was used in all tests.

RESULTS

In the first test an attempt was made to maintain temperatures of 100°, 105°, 110°, and 115° F in the heating boxes. Because of the small size of the boxes in relation to the roots, it was 17 hours before the roots came up to the proper temperature. Total exposure time was 41 hours. The results of the first test are given in Table 1. Nematodes were killed at 110° and 115°.

Table 1. Presence of viable root-knot nematodes in five sweetpotato roots before and after dry heat treatment and number of roots viable after heating. Roots were heated for a total of 41 hours.

Root temperature °F for 24 hours	Number of roots assaying positive for nematodes		Number of roots producing slips
	Before heating	After heating	
100	4	2	2
105	5	1	2
110	4	0	0
115	5	0	0

Nematodes were also reduced in number at 100° and 105° but control was not complete. Two of the five roots survived the treatment at 100° and 105° but all roots were killed at 110° and 115°. The results of this test suggested that temperatures of 110° and 115° for 24 hours were sufficient to kill nematodes, but that seed roots severely infected by nematodes and dry rot would not survive this treatment. In subsequent tests clean roots were heated along with nematode infested roots in order to determine the influence of heating on survival and slip production.

Average oven and root temperatures of 102°, 105°, 106° and 110° F were maintained for a minimum of 9 hours in the second test. Total exposure time was 27 hours. Results are shown in Table 2. Root temperatures as high as 110° for a minimum of 9 hours was not sufficient to

Table 2. Viable root-knot nematodes in five sweetpotato roots before and after dry heat treatment and average number of slips produced by five healthy roots after treatment.

Root temperature °F for 9 hours	Number of roots assaying positive for nematodes	Average number of nematodes	Number of roots viable	Average number of slips per root
102 Preheat	5	92	-	-
Heated	5	33	5	14
105 Preheat	5	83	-	-
Heated	3	20	4	17
106 Preheat	4	79	-	-
Heated	3	4	4	17
110 Preheat	5	74	-	-
Heated	4	31	5	13

kill the eggs within the roots, although the numbers were greatly reduced. At least 80% of the roots survived the heating, and slip production was satisfactory.

In the third test six nematode-infected roots and 10 clean roots were heated in a large drying oven at 108° F for a total of 27 hours. It took 3 hours to raise the temperature of the roots to 108°. Results are shown in Table 3. Only one of the six infected roots contained viable

nematodes after heating. Nine of the 10 clean roots were capable of producing slips after treatment.

Table 3. Viable root-knot nematodes in six infected sweetpotato roots before and after dry heat treatment and average number of slips produced by 10 healthy roots after treatment.

Treatment	Number of roots assaying positive for nematodes	Average number of nematodes per root	Number of roots viable	Average number of slips per root
None	6	144	10	-
108° F for 27 hours	1	2	9	24

Table 4. Viable root-knot nematodes in 28 infected sweetpotato roots before and after dry heat treatment and average number of slips produced by 19 healthy roots after treatment.

Treatment	Number of roots assaying positive for nematodes	Percentage of heated roots viable	Average number of slips per root
None	28	-	-
113° F for 36 hours	0	68	20

Twenty-eight infected roots and 19 clean roots were heated at 113° F for a total of 36 hours in the fourth test. The time required for the roots to warm up to 113° is illustrated graphically in Figure 1. The root temperature increased to 108° within 3 hours, as in the previous test, but required another 4 hours of heating to increase to 113°.

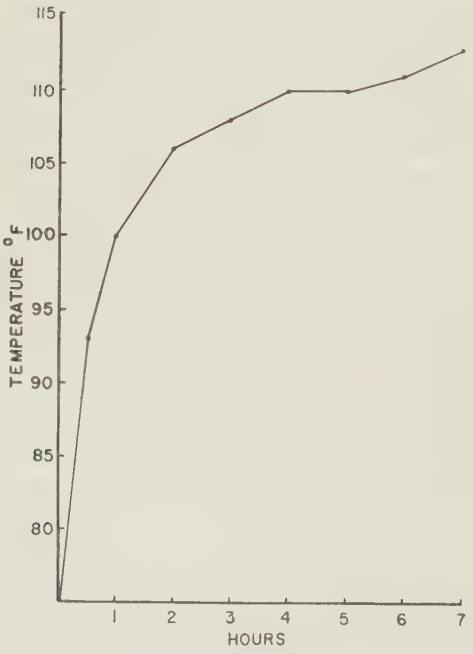

FIGURE 1. Time required for the temperature of a sweetpotato seed root to reach oven temperature when dry-heat treated at 113° F.

Control of nematodes and production of slips by surviving roots is reported in Table 4. No nematodes survived this treatment. Sixty-eight % of the clean roots were still viable after treatment and produced an average of 20 slips per root after being bedded for 4 weeks.

DISCUSSION

Although the minimum time-temperature combination for 100% control of root-knot nematodes within sweetpotato seed roots was not established, the results from the several tests indicated that a root temperature of 110° to 113° F for a minimum of 24 hours is necessary. In the first test a root temperature of 110° for 24 hours was sufficient for 100% control. In the third test one root contained viable nematodes after being heated to 108° for 24 hours. Sound sweetpotato roots free of dry rot and other obvious injuries are surprisingly tolerant of the treatment as indicated by the production of slips by 68% of the roots heated to 113° for 29 hours.

In order to properly treat roots a large source of heat is necessary as well as adequate circulation. Temperature control must be critical, as overheating could be serious.

The results of these preliminary tests using a dry heat treatment to eliminate root-knot nematode from sweetpotato seed roots suggest that the method has possibilities both for sweetpotato growers and possibly for other heat tolerant propagating stocks.

Several growers in San Bernardino County have used their curing rooms to dry heat treat their sweetpotato seed roots. Slip production following treatment has been satisfactory.

TARRAGON

Tarragon, Artemisia dracunculus, an aromatic herb is grown on a small acreage in the United States. The plant is a perennial and the foliage is processed and marketed for use in seasoning food.

In April of 1957, a planting of tarragon in San Jacinto, California was found to be severely infected with the root-knot nematode Meloidogyne hapla. The planting was approximately 5 years old and the crowns of the plants were dying out. New growth surrounding the crowns was weak and unthrifty.

The grower wished to propagate a new planting on clean ground, but had no nematode-free propagating stock available. Tarragon is normally propagated by using new shoots which arise from hard, horizontal rhizomes. An attempt was made to eradicate the root-knot nematodes from the rhizomes using hot water treatments.

Goheen and McGrew (3) tested a number of time-temperature combinations for the control of Meloidogyne hapla and Pratylenchus penetrans on strawberry. They obtained satisfactory control of the nematodes without killing the plants at 127° F for 2 minutes. They also obtained nematode kill at other time-temperature combinations ranging from 6 seconds at 150° to 7 minutes at 121°. Lear and Lider (4) obtained control of root-knot nematodes on grape rootings at 118° for 30 minutes.

MATERIALS AND METHODS

The following time-temperature combinations were used to treat rhizomes in a hot water bath: 113° F for 25 and 30 minutes, 116° for 10, 15 and 20 minutes. Ten nematode-infected rhizomes were treated at each time-temperature combination. After treatment the rhizomes were potted in a sandy loam soil along with a tomato seedling. The tomato seedling was used to index for survival of the root-knot nematode in those cases in which the tarragon rhizomes did not survive the treatment. Ten non-treated rhizomes were potted in clean soil as checks.

One further lot of 54 rhizomes was hot-water treated for 25 minutes at 113° F. After treatment the rhizomes were potted individually in a sandy loam soil.

RESULTS

In the first test all the non-treated rhizomes survived and produced plants. Eighty % of these were found to be infected with root-knot nematodes. The highest survival in the treated material was 50%. None of the surviving rhizomes were found to be infected. However, tomatoes grown in soil with the non-surviving rhizomes were found to be infected in a few cases. Nematodes survived on one rhizome treated at 113° F for 30 minutes and one at 116° for 20 minutes (Table 5).

Table 5. Hot water treatment for control of root-knot nematode in propagating stock of tarragon.

Temperature (° F)	Time period (in minutes)	Number of rhizomes treated	Number of rhizomes surviving	Number of surviving rhizomes showing root-knot nematode galling	Number of dead rhizomes on which root-knot nematodes survived
---	Control ---	10	10	8	-
113	25	10	5	0	0
113	30	10	4	0	1
116	10	10	5	0	0
116	15	10	3	0	1
116	20	10	4	0	2

Of the 54 rhizomes hot-water treated at 113° F for 25 minutes 37, or 68%, survived. Of these surviving, 15 showed root-knot nematode galling when examined later. The rapid production of lateral shoots from these rhizomes made it possible to propagate 50 plants from the 22 original rhizomes surviving treatment and free of nematodes only 2 months after treatment.

The propagation of clean plants was also attempted by making cuttings of new shoots arising from infected rhizomes and potting them in soil. Of 18 cuttings propagated, five showed root-knot nematode galling when examined after 2 months.

Neither the hot water treatment nor the propagation of new shoots in clean soil were completely successful in producing nematode-free plants. Approximately 60% of the surviving hot-water treated rhizomes were free of nematodes, whereas 70% of the plants produced from new shoots were free of root knot. In any case, if each shoot is potted separately the nematode-free plants can be selected and used as a source of clean propagating stock.

The use of higher temperatures to completely kill all nematodes in the rhizomes appears to be limited by the ability of the rhizomes to survive the treatment.

As far as the authors are aware, this is the first report of Artemisia dracunculus as a host of Meloidogyne hapla.

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EFFECT OF BARLEY STRIPE MOSAIC ON WHEAT[1]

Paul J. Fitzgerald and R. G. Timian[2]

Barley stripe mosaic is a potentially serious disease of wheat and barley, according to several workers. McKinney (6) and Eslick (2) reported that the disease is caused by a seed-borne virus. Transfer of the virus from diseased to disease-free plants was described by McKinney (7), Gold et al. (4), and Fitzgerald et al. (3). Army and McNeal (1), reported that the uptake of some plant nutrients is seriously affected by barley stripe mosaic. The effect of the disease on spring wheat was reported by McNeal et al. (8) and Hagborg (5).

Since information concerning the virus in winter wheat is meager or lacking, this investigation was undertaken to determine to what extent winter wheat varieties are affected by the barley stripe mosaic virus.

EXPERIMENTAL PROCEDURE

Stripe mosaic virus-infected seed lots of Wasatch, Cache, Turkey, Columbia, and Itana winter wheats were obtained by inoculating plants of each of the varieties in the field in 1957 with expressed sap from diseased barley plants grown in the greenhouse.

An assay of the seed thus obtained was made in the greenhouse at North Dakota State College, Fargo, North Dakota. Infection percentages are shown in Table 1.

Table 1. Effects of barley stripe mosaic virus on the performance of five varieties of winter wheat grown under sprinkler irrigation at Aberdeen, Idaho, 1957-58[a].

Variety and type of seed	Yield[b] per acre (bushels)	Weight per bushel (pounds)	Height (inches)	Date of first heading	Infection percentage in	
					Planted seed	Harvested grain
Wasatch						
Diseased	50.3	60.8	43	May 30	55	33
Disease-free	58.1	61.3	44	May 29	0	0
Cache						
Diseased	42.9	59.1	42	June 2	59	18
Disease-free	57.9	61.1	43	May 31	0	0
Turkey						
Diseased	51.4	59.7	42	May 30	56	28
Disease-free	61.4	60.7	42	May 29	0	0
Columbia						
Diseased	52.6	60.1	38	May 28	60	34
Disease-free	69.2	60.7	38	May 28	0	0
Itana						
Diseased	54.7	60.4	40	May 30	70	41
Disease-free	64.4	60.5	41	May 30	0	0

[a]Each value is the average from six replications.
[b]L.S.D. (.05) for varieties = 6.36 bushels; L.S.D. for mean differences -- diseased versus disease-free = 4.49 bushels; Coefficient of variability = 14.26%.

Diseased and disease-free seed of the five varieties of winter wheat were grown under sprinkler irrigation in paired 4-row plots at the Aberdeen Branch Experiment Station in 1957-58. Fourteen feet of the two center rows of each plot were harvested for yield. Additional samples were harvested from disease-free plots of each variety to ascertain the degree of spread of the virus.

Samples were submitted to the Western Regional Quality Laboratory, Pullman, Washington, for milling and baking tests.

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RESULTS

Significant yield reductions (Table 1) occurred in all varieties infected with the virus, ranging from a 7.8-bushel difference between healthy and infected Wasatch to a 16.6-bushel difference in Columbia. The mean yield of all diseased plots was 50.4 bushels per acre, while disease-free plots yielded 62.2 bushels. This 11.8-bushel decrease between the means of diseased and disease-free plots represents an average reduction of almost 19%. Individual varietal reductions ranged from 13.4% for Wasatch to 25.9% for Cache.

Protein content was slightly higher in samples from diseased plots, but preliminary results indicate that the milling and baking qualities of winter wheat infected with the barley stripe mosaic virus are not significantly different from those of disease-free lots of the same variety grown under comparable conditions.

The assay for virus (Table 2) in seed from disease-free plots indicates either that the virus had not been transferred from the adjacent diseased plots or that the transfer occurred so late in the development of the plants that the virus was not contained in the kernels of the plants in the disease-free plots.

Table 2. Results from an assay for virus in the harvested seed lots of five winter wheats grown at Aberdeen, Idaho, 1957-58.

Variety, type of seed, and row designation[a]	Plants	
	Grown (number)	Infected (%)
Wasatch		
Diseased, 1	201	33
Disease-free, 1a	281	0
Disease-free, 1a1	271	0
Disease-free, 1a4	278	0
Columbia		
Diseased, 2	277	34
Disease-free, 2a	290	0[b]
Disease-free, 2a1	250	0
Disease-free, 2a4	248	0
Turkey		
Diseased, 3	291	28
Disease-free, 3a	272	0
Disease-free, 3a1	254	0
Disease-free, 3a4	249	0
Cache		
Diseased, 4	192	18
Disease-free, 4a	243	0
Disease-free, 4a1	267	0
Disease-free, 4a4	272	0
Itana		
Diseased, 5	313	41
Disease-free, 5a	324	0
Disease-free, 5a1	250	0
Disease-free, 5a4	275	0

[a]1a is a composite sample from yield rows. 1a1 refers to the first disease-free row of Wasatch adjacent to the diseased plot. 1a4 refers to the fourth row, that is, this row was separated from the diseased plot by three other disease-free rows. The a, a1, and a4 designations have the same meaning for the other four varieties.

[b]No virus recovered from seemingly infected plant.

Results from a subsequent greenhouse test at Fargo, North Dakota indicate that under conditions of close row spacing (2 inches) and periodic air movement (from electric fan), the virus can be transferred from diseased Manchuria barley and Wasatch winter wheat to disease-free Stewart durum spring wheat, Lee hard red spring wheat, and disease-free Manchuria barley.

Diseased rows in this test were flanked on one side by eight disease-free rows and on the other side by five disease-free rows. Infection was present in the most distant rows from the source of infection in all varieties.

The hard red spring wheat showed a higher percentage infection when Wasatch was used as a source of the virus, and the barley infection was greater when diseased barley was the source of the virus.

DISCUSSION AND CONCLUSIONS

Yield reductions were considerably smaller than had been anticipated after spring observations of severe foliar symptoms, reduced stands, and reduced vigor. The average yield reduction of almost 19% in diseased plots compares with an average reduction of 32.8% in eight varieties of spring wheat (8) and a reduction of 31% in barley (2). Apparently some of the effects of the disease were offset by the vigorous growth of the healthy plants in a row, which were grown under conditions of reduced competition. Probably most of the crop was produced by the 30 to 45% of disease-free plants.

The lack of transfer of the virus from diseased to disease-free plants in the field requires further study in light of the results of the greenhouse test at Fargo, North Dakota, in which the virus was transferred to all rows of durum and hard red spring wheats and to Manchuria barley.

A level of virus infection of 27% was obtained from the transfer of the virus under natural conditions from diseased Trebi barley to disease-free Wasatch and Cache wheats in 1957 (3). In that study the barley was seeded in the same row with transplanted wheat seedlings; therefore, transfer may have occurred at an early stage. In the present field study plants were grown in rows spaced 12 inches apart. As a result, leaf contact would occur at a later stage of plant growth in the spaced rows than when disease-free plants were placed in an infected row. The fact that no infection was present in the seed from spaced rows adjacent to diseased plots in the present study indicates either that the older plants are resistant to infection by the virus or that infection takes place but the virus does not invade the kernels.

If virus transfer from diseased to disease-free winter wheats under field conditions is infrequent, then a rapid spread of the disease is not likely to occur. Furthermore, if the level of infection remains low under natural conditions the economic importance of the disease may be negligible.

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THE RING SPOT DISEASE OF RAPE IN AN INLAND PARKLAND REGION

T. C. Vanterpool[1]

Abstract

Ring spot of crucifers, hitherto reported as restricted to coastal and other moist regions of the world, was found on summer oil rape (Brassica campestris L. var. annua Reichb.) in an inland area with an annual precipitation of 15 inches. About half of this falls during the growing season. The causal fungus, Mycosphaerella brassicicola (Fr.) Lindau, was isolated from plant debris after harvest and from diseased seeds. The latter finding is additional evidence in support of its seed-borne nature. The present report is the first of ring spot on crucifers in Saskatchewan and on rape in Canada. The disease was probably brought into the province on the Polish variety by immigrants from Europe.

Mycosphaerella brassicicola (Fr.) Lindau was collected on September 4, 1958, on the stubble of oil rape (Brassica campestris L. var. annua Reichb.) in several fields in the black soil zone of east-central Saskatchewan a few days after harvest. This is the first report of ring spot on crucifers for the province, and on rape for Canada. In view of the reported restriction of ring spot of crucifers to coastal and other moist regions of the world (2, 3) and the conflicting evidence on the seed-borne nature of the disease (2, 5, 6), its presence in an inland subhumid region, with an average annual precipitation of 15 inches, is of more than local interest. The recent comprehensive paper by Nelson and Pound (2) on the relation of environment to ring spot of crucifers makes publication of this report on the finding of the disease in an inland region advisable.

The disease was widely scattered throughout the infested fields. In some areas the stubble showed more than 50% infection; the higher disease rating being found in second crop rape. The early maturing Polish variety, originally brought in by European immigrants, is commonly grown in the district, where rape has been used as a cash crop for about 10 years. The currently-used seed is grown locally. It seems probable that the disease has been present in the district for some time and has passed unnoticed.

The lesions were slaty gray to dark slaty gray, speckled with numerous, closely-packed, black spermogonia; no perithecia were found. The central portion of a few of the older lesions were somewhat bleached. The lesions ranged in size from a quarter of an inch to four inches in length and often encircled the stem, all parts of which were liable to attack. The seed pods were also attacked. No leaves were available for examination.

It was not possible to culture the fungus by spore dilution methods, as the spermalia did not germinate. Strips of the bark with numerous spermogonia were dissected into very fine pieces in 1-ml portions of sterile water. Loopfuls of this inoculum were distributed in Petri dishes into which various media were than poured. Usually a few colonies of the slow-growing fungus were obtained in each Petri dish. Many of these arose from mycelium of the outer part of the spermogonial wall. Many cultures were obtained by plating seed taken from infected pods immediately after harvest, but the fungus was also isolated from seed of diseased pods which had been kept in the laboratory in a paper bag for 15 months. Because of the slow-growing nature of the fungus in culture, the seedlings arising from infected seed in Petri dishes were only slightly infected. Seed infection did not appear to be deep-seated, since the seeds which gave rise to M. brassicicola colonies germinated reasonably well. The symptoms on stems and pods, the measurements of spermogonia and spermatia, and the cultural characters of the fungus are in agreement with published information (2, 4, 6) on ring spot, and leave no doubt as to the identity of the disease.

The locality in which the disease was found is situated about 75 miles east of Saskatoon in latitude 52N and longitude 105W, and several miles within the parkbelt. It is immediately southeast of Lake Lenore, which is 35 square miles in area. The topography is flat and the cultivated fields are usually large, but there are many groves of trees scattered over the landscape. These trees give considerable protection from the winds sweeping across the prairie, so that although the annual precipitation is virtually the same as on the prairie 100 to 300 miles to the southwest, the evaporation is considerably reduced, humidity is higher, and crops can make

[1]Supported by a research grant from the Saskatchewan Agricultural Research Foundation.

more efficient use of the available soil moisture. It is generally felt that dews are more frequent and heavier, although official records on dews are not available. The average annual precipitation for the district is 15 inches, of which about 8 inches fall as rain from May to August inclusive, when the rape crop is growing. The long-time average maximum and minimum daily temperatures (° F) and the average rainfall in inches for the four summer months, as well as corresponding records for 1958, are given in Table 1.

Table 1. The long-time and 1958 temperature (°F) and rainfall data for May to August inclusive, in east-central Saskatchewan.

	Long-time averages				1958 averages			
	Daily temperature means			Rainfall	Daily temperature means			Rainfall
Month	max.	min.	mean	(in inches)	max.	min.	mean	(in inches)
May	64	39	51	1.35	70	41	55	0.45
June	71	47	59	2.56	74	44	59	0.39
July	79	53	66	2.38	77	52	64	3.80
August	76	50	63	1.77	79	51	65	0.25

When the data in Table 1 are compared with those presented by Nelson and Pound (2), it will be seen that the monthly mean temperatures (°F) for the growing season, where the disease occurred, are within the range of those in the Pacific Coast regions where ring spot of cabbage is prevalent. The minimum nightly temperatures are probably lower than in the Pacific Coast region. Such low temperatures on clear, still nights would explain the heavy dews sometimes experienced. However, abundant moisture, as rainfall, cannot be coupled with the moderate temperatures in the Saskatchewan region.

The fact that the summer of 1958 was one of the driest on record, with a rainfall of less than 5 inches, gives added interest to the relation of the weather to the outbreak of ring spot. In view of the scarcity of observations on the disease under Saskatchewan conditions, a discussion of how the local environment affects the disease would be largely speculative. However, the heavy rainfall of July 12 and 13 would appear to have been responsible for the crucial ascospore discharge and spread of the disease. Infested stubble appeared to be a serious source of primary inoculum since rape grown in successive years in the same field was most heavily attacked, followed in disease incidence by fields adjoining old rape stubble. Some abnormalities concerning outbreaks of this disease in Ireland were pointed out by McKay (1). He reported that on some farms where large-scale cultivation of vegetables was a regular practice, ring spot was something of a problem every year irrespective of weather conditions, and also that many such places were situated in the driest part of the country.

It is suggested that the disease was brought into the province initially either on infected seed or on bits of infected stem or leaf material mixed with the seed. Another possibility is that it arose from indigenous cruciferous weeds. The disease is now established in the Lake Lenore area, where infected seed and especially stubble serve as sources of primary inoculum. Despite the low annual precipitation for the area, the conditions otherwise provided by the parkland environment, such as the relatively low evaporation rates and the snow cover and cessation of growth during five, very cold, winter months, are sufficient to permit the establishment of the disease in this northern inland location.

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DEPARTMENT OF BIOLOGY, UNIVERSITY OF SASKATCHEWAN, SASKATOON, CANADA

PHYTOPATHOGENIC AND SAPROPHYTIC FUNGI ASSOCIATED WITH FORAGE LEGUME SEED[1, 2]

Charles M. Leach[3]

Summary

The nature and frequency of seed-borne fungus pathogens associated with a total of 1300 seed lots of eight species of forage legumes (Pisum sativum var. arvense, Medicago sativa, Trifolium hybridum, T. incarnatum, T. pratense, T. repens, Vicia sativa and V. villosa) were determined by the malt agar plating method. Pathogens most consistently isolated were: Mycosphaerella pinodes and Botrytis cinerea from field pea seed; Phoma herbarum var. medicaginis, Fusarium roseum and Stemphylium botryosum from alfalfa seed; B. cinerea, F. roseum, Phoma trifolii and S. botryosum from alsike clover seed; B. cinerea, F. roseum, P. trifolii, Sclerotinia sclerotiorum and S. botryosum from crimson clover seed; and Ascochyta pisi from hairy vetch seed. A number of other pathogens were isolated much less frequently. Seed of red clover, ladino clover, and common vetch were relatively free of pathogenic fungi. In addition to pathogens, 24 genera of saprophytic fungi were isolated; of these 10 were common to all eight species of forage legumes.

The quality of certified seed sold in commerce is commonly determined by a combination of field certification and laboratory examination. The major measures of seed quality as determined by seed testing laboratories are viability, varietal purity, and freedom from weed and other crop seeds. Another indication of quality is the "health" of the seed, that is its freedom from seed-borne phytopathogens. This latter measure of seed quality has received little emphasis in the United States although it is well known that many plant pathogens are seed-borne (12).

The purposes of this study were to assess the "health" of Oregon-produced forage legume seed by determining the nature and frequency of seed-borne pathogens and saprophytes, and to develop methods for detecting the presence of seed-borne pathogens applicable to routine service testing in seed testing laboratories. The second objective of this study has not been completed and is not included in this report.

LITERATURE

Records of the occurrence of plant pathogens with forage legume seeds in Oregon are few. Hardison reported Botrytis anthophila associated with red clover seed (6) and Sclerotina spermophila with seed of ladino clover (5). Leach has noted the occurrence of seed-borne organisms associated with clover (8, 9, 10) and other forage legume species (11). An annotated list of the seed-borne pathogens of forage legumes is included in the compilation by Noble, deTempe, and Neergaard (12).

PROCEDURE

Samples from 1300 seed lots were obtained mainly from seed mailed to the Oregon Seed Cooperative Testing Laboratory for routine purity and germination tests. In addition, a few samples were obtained from commercial seed testing laboratories. The species of legumes surveyed were alfalfa (Medicago sativa), field pea (Pisum sativum var. arvense), red clover (Trifolium pratense), crimson clover (T. incarnatum), ladino clover (T. repens), alsike clover (T. hybridum), common vetch (Vicia sativa) and hairy vetch (V. villosa). Table 1 shows the number of seed lots examined from each species. All samples were examined within 9 months following harvest. Sub-samples taken from the seed laboratory samples were placed in a mixer[4] and rotated for 1 minute at 35 rpm to mix the seed thoroughly. The mixer and associ-

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[1]Approved for publication as Technical Paper No. 1301 of the Oregon Agricultural Experiment Station.
[2]Work supported by Regional Project WM-35, "Facilitating the Marketing of Seed Through Improved Testing Procedures."
[3]Assistant Plant Pathologist, Oregon Agricultural Experiment Station.
[4]Copy of the "p-k twin shell laboratory blender" manufactured by the Patterson Kelly Company Incorporated, East Stroudsburg, Pennsylvania.

Table 1. Numbers of seed lots sampled for seed-borne pathogens.

Legume	1956	1957	1958	Total
Alfalfa		60	95	155
Field pea		101	92	193
Alsike clover			112	112
Crimson clover		44	170	214
Ladino clover			82	82
Red clover			136	136
Common vetch			71	71
Hairy vetch	104	101	132	337
Total	104	306	890	1300

ated equipment were cleaned with 95% ethanol after mixing each sample to prevent contamination of subsequent samples by pathogens or by troublesome saprophytic fungi. With the use of a vacuum counter (7), 200 seeds from each sample were placed on 20 ml of 2% malt extract agar (pH 5-6) in 100-mm diameter Petri plates. Thirteen seeds plated on each of 15 plates, and five seeds on one plate, accommodated each sample. The plates were placed within transparent polyestyrene containers and incubated at room temperature (70° to 80° F) in diffused daylight. After 10 days the plates were examined both macroscopically and microscopically (20X) for the presence of pathogens.

All procedures used for the detection of seed-borne plant pathogens are selective to some degree. The plating method utilized in this study is no exception, and though suitable for detecting many seed-borne fungus pathogens of forage legumes, it is unsuitable for detecting seed-borne viruses, nematodes, obligate parasitic fungi, and fungi unable to grow well on malt extract agar.

RESULTS AND DISCUSSION

Data concerning the occurrence of pathogens are presented in Tables 2 and 3. Table 2 is a comparison of the distribution of pathogenic species in the different legumes for the years 1957 and 1958. Table 3 relates the occurrence of saprophytes. The pathogens considered most important as factors affecting the quality of Oregon's forage legume seed are briefly as follows:

1. Field pea (Table 2): Twelve pathogenic fungi were associated with field pea seed. Only two species occurred frequently enough to merit consideration as possible factors affecting the quality of this seed. Most important was Mycosphaerella pinodes which infested 55% of the 1957 seed lots and 88% of the 1958 seed lots, and which annually causes severe losses in peas grown in western Oregon. Botrytis cinerea, an omnivorous pathogen, was also encountered fairly frequently. Although B. cinerea is not important in Oregon fields, it can be a virulent pathogen during germination tests in the seed laboratories where it spreads rapidly, damps-off seedlings, and directly affects the accuracy of tests.

2. Alfalfa (Table 2): Seven pathogenic fungi were isolated from alfalfa seed, however only three species were observed frequently. Most common was Stemphylium botryosum, a fairly important pathogen in certain temperate zones (2, 13), but of minor importance in Oregon. Second in order of frequency was Phoma herbarum var. medicaginis (=Ascochyta imperfecta), a fungus universally recognized as a major pathogen of alfalfa. Fusarium roseum was the third most frequent pathogen. The pathological significance of F. roseum is uncertain, for although it has been proven to be an active root pathogen of alfalfa in certain regions of the United States[5], it has never been identified in this role in Oregon. All the pathogens mentioned damp-off seedlings in seed laboratory germination tests and thus affect the accuracy of the tests.

3. Alsike clover (Table 2): The pathogens isolated most frequently from alsike clover were Botrytis cinerea, Fusarium roseum, Phoma trifolii, and Stemphylium botryosum. B. cinerea, though not recognized as an important pathogen of clover in the field, does attack seedlings in the soil and during seed laboratory germination tests. The pathological significance of the association of F. roseum with alsike clover seed has not been determined by the writer but the fungus rapidly kills seedlings in blotter type germination tests. Corden (4) found that F. roseum will cause a severe cortical root rot of alsike clover in Oregon. P. trifolii and S. botryosum are well known clover pathogens which are generally of minor importance to field stands of alsike clover in Oregon, but do kill seedlings during germination tests.

[5]Information obtained through personal correspondence.

Table 2. Pathogenic fungi associated with forage legume seed[a].

Legume	Pathogen	% seed lots infested[b]		% level of infestation[c]			
				Average		Highest recorded	
		1957	1958	1957	1958	1957	1958
Alfalfa	Botrytis cinerea		1		1		1
	Fusarium oxysporum	2	3	1	2	1	3
	F. roseum	2	22	1	1	1	3
	F. spp.	8	13	1	1	1	1
	Phoma herbarum v. medicaginis	27	34	1	1	3	3
	Phoma trifolii	5	3	1	2	3	3
	Stemphylium botryosum	42	77	3	8	9	37
Field pea	Ascochyta pinodella	3		1		1	
	A. pisi		2		1		1
	Botrytis cinerea	17	22	1	1	2	4
	Colletotrichum sp.	1		1		1	
	Fusarium oxysporum	1		1		1	
	F. roseum	1	4	1	1	1	1
	F. tricinctum	5		1		1	
	F. spp.	6	11	1	1	1	1
	Mycosphaerella pinodes	55	88	1	4	5	15
	Pythium sp.		1		1		1
	Sclerotinia sclerotiorum	1		1		1	
	Stemphylium botryosum		3		1		2
Alsike clover	Botrytis cinerea		80		3		11
	Fusarium roseum		30		2		8
	F. spp.		13		1		2
	Phoma trifolii		41		2		9
	Stemphylium botryosum		34		1		8
	Verticillium albo-atrum		2		1		1
Crimson clover	Botrytis cinerea	59	45	1	2	3	5
	Fusarium roseum	66	74	1	9	3	63
	F. oxysporum		1		1		1
	F. tricinctum		1		1		1
	F. spp.		12		2		4
	Kabatiella caulivora						
	Phoma trifolii	96	59	2	3	9	14
	Sclerotinia sclerotiorum						
	(a) mycelium	11	12	1	1	2	2
	(b) sclerotia[d]	8	18	-	-	-	-
	Stemphylium botryosum	89	67	3	7	25	32
	Helminthosporium tetramera	31	31		1		4
Red clover	Ascochyta pisi		2		1		1
	Botrytis anthophila		12		1		5
	B. cinerea		2		1		1
	Fusarium spp.		1		3		3
	Helminthosporium tetramera		2		1		1
	Phoma trifolii		4		1		1
	Stemphylium botryosum		11		1		2
Ladino clover	Botrytis cinerea		6		1		1
	Fusarium roseum		13		3		8
	F. spp.		7		1		1
	Phoma trifolii		12		2		7
	Sclerotinia spermophila		1		1		1
	Stemphylium botryosum		17		1		5
	S. trifolii		10		1		2
Common vetch	Ascochyta pisi		14		1		3
	Botrytis cinerea		9		1		1
	Helminthosporium tetramera		1		1		1
	Fusarium roseum		9		1		1
	F. spp.		18		1		3
	Stemphylium botryosum		7		1		2

		% seed lots infested[b]			% level of infestation[c]					
					Average			Highest recorded		
		1956	1957	1958	1956	1957	1958	1956	1957	1958
Hairy vetch	Ascochyta pisi	68	30	84	3	1	4	9	3	36
	A. sp.	3			2			1		
	Botrytis cinerea	5	7	6	1	1	1	2	1	2
	Fusarium roseum			2			1			1
	F. spp.			2			1			1
	Helminthosporium tetramera			12			1			1
	Pythium spinosum			1			1			1
	Sclerotinia sclerotiorum	3			1			1		
	Stemphylium botryosum			16			1			2

[a]All percentages are to the nearest integer.

[b]Numbers of seed lots sampled shown in Table 1.

[c]Level of infestation is the percentage of the sample of 200 plated seeds infested with a pathogen.

[d]Sclerotia data were obtained from purity analyses conducted by the Oregon Cooperative Seed Testing Laboratory in which 489 samples were analyzed in 1957 and 427 in 1958.

Table 3. Saprophytic fungi associated with seed of eight species of forage legumes[a].

Saprophyte	Medicago sativa	Pisum sativum var. arvense	Trifolium hybridum	T. incarnatum	T. pratense	T. repens	Vicia sativa	V. villosa
Acremoniella atra								++
Alternaria tenuis	++++	+++	++++	++++	++++	++++	++++	++++
Aspergillus spp.	++	+++	+++	++	++	+++	++	+++
Candida sp.					+			
Cephalosporium sp.				+	+			
Cephalothecium sp.	+	+		+	+			
Chaetomium spp.	+			+	+			+
Cladosporium cladosporioides	++++	++++	++++	++++	++++	++++	+++	++++
Cladosporium elatum	++	+	+++	+	+++	+++		++
Epicoccum nigrum	++	+	++++	++	++	++	+	+++
Fusidium sp.								+
Geotrichum sp.					+			
Hormiscium sp.					+			
Mucor spp.	+++	++	+++	+++	++	+++	++	++
Neurospora sp.								+
Nigrospora oryzae				+				
Papulospora sp.								+
Penicillium spp.	++++	+++	+++	+++	++++	+++	+++	+++
Pullularia pullulans	++	+	++	++	++	+	+	+++
Rhizopus stolonifera	++	+++			++		+++	+++
Scopulariopsis brevicaulis					+	++		
Stemphylium consortiale	++	+	++	+++	+++	+++	+	+++
Trichothecium sp.								+
Trichoderma sp.	+	+			++			+

[a]++++ = very frequent; +++ = frequent; ++ = occasional; and + = rare.

4. Crimson clover (Table 2): Seed lots of crimson clover showed a greater frequency of seed-borne pathogens than did any of the other legumes. B. cinerea, F. roseum, P. trifolii and S. botryosum were commonly associated with this seed. Helminthosporium tetramera, a recognized root pathogen of certain grasses but not of legumes, was encountered for the first time[6]. The significance of the presence of H. tetramera with crimson clover seed has not been determined. Fewer seed lots were infested with Sclerotinia sclerotiorum (=S. trifolium) than with the previously mentioned fungi. However as a major pathogen of clover it cannot be ignored, for in some years it causes serious losses in Oregon. Although S. sclerotiorum was more usually present as sclerotia mixed with seed, mycelial infection of seeds was not uncommon (10). Also of interest was the presence of Kabatiella caulivora with crimson clover seed. This is only the second[7] time that the natural occurrence of this organism with seed has been reported. The frequency of this fungus is not included in Table 2 because it was recognized only after the majority of crimson clover seeds had been examined. Culturally, K. caulivora is somewhat like the dark forms of Pullularia pullulans and it is possible that K. caulivora was unknowingly misidentified as P. pullulans. In western Oregon K. caulivora is potentially a serious pathogen and in 1959 it devastated a number of fields of crimson clover. Circumstantial evidence indicated that these epiphytotics were initiated through seed-borne inoculum.

5. Red clover (Table 2): Red clover seed lots were the least infested by seed-borne pathogens of any of the seed examined. The seven pathogenic species isolated were all present at

[6]H. tetramera is not reported as a pathogen of clovers in the literature, nor is there any mention of it in association with clover seed.

[7]Dr. Mary Noble (Edinburgh, Scotland) observed K. caulivora to be seed-borne in Oregon-produced crimson clover during the 1959 international referee tests conducted by the Plant Disease Committee of the International Seed Testing Association.

low levels. Botrytis anthophila and S. botryosum were most frequently isolated, but they only infested 11% of the seed lots. B. anthophila systemically infects clover plants and prevents anthesis at flowering; however, it has never been shown to be a serious problem in the United States and has been recorded only in the State of Oregon (6). B. anthophila does not adversely affect seed laboratory germination tests. The significance of S. botryosum has been discussed previously.

6. Ladino clover (Table 2): Seed lots of ladino clover were very lightly infested with pathogens. S. botryosum, which infested only 17% of the seed lots, was most frequently isolated. Of the six pathogens isolated infrequently, Sclerotinia spermophila and Stemphylium trifolii were unique in that they were associated only with ladino clover.

7. Common vetch (Table 2): Seed lots of common vetch were very lightly infested with pathogens. None of those observed are considered important as factors affecting the quality of common vetch seed when present at such low levels.

8. Hairy vetch (Table 2): Six pathogens were isolated from hairy vetch seed. Only Ascochyta pisi was present fairly often. The number of seed lots infested with A. pisi varied considerably from year to year; 84% of the samples were infested in 1958 and only 30% in 1957. The level of infestation within seed lots also varied greatly. In 1956 and 1957, 95 and 100% of the infested seed lots showed less than 2.5% of the seed infested, whereas in 1958 50% of the seed lots had levels of infestation higher than 2.5%. A. pisi is usually of minor importance in Oregon, except during cool and moist springs when it may cause considerable damage. In other regions it is reported as a limiting factor in seed production (1, 14). A. pisi will kill infected seedlings during germination tests and thus affect the accuracy of the test.

9. Saprophytic fungi (Table 3): Twenty four genera of saprophytic fungi were isolated from seed of the eight leguminous species. Common to most of the legumes were Alternaria tenuis, Aspergillus spp., Cladosporium cladosporioides, C. elatum, Epicoccum nigrum, Mucor spp., Penicillium spp., Pullularia pullulans, Rhizopus stolonifera and Stemphylium consortiale. In general these microorganisms are not known to affect the germination and development of field-sown seed, however they cannot be completely ignored for several reasons: a) Pathologists investigating seed-borne pathogens must distinguish between saprophytic and parasitic species, particularly where a saprophyte may be mistakenly identified as a pathogen or vice versa. b) Several of the saprophytic fungi listed can behave as weak to virulent pathogens in the seed testing laboratories' germination tests. When seedlings are attacked by these organisms, the accuracy of germination tests may be affected. Rhizopus stolonifera is such an organism; it may spread rapidly through a variety of germination test substrates, killing seedlings in its path. Cladosporium cladosporioides is another common seed-borne saprophyte of forage legumes which sometimes parasitizes red clover seedlings in germination tests. Similarly, strains of Alternaria tenuis will often attack seedlings in germination tests. c) Certain rapidly growing saprophytes tend to overgrow and mask the presence of pathogens in the media plating method; R. stolonifer, in particular. Alternaria tenuis is less important. d) Certain of the saprophytic species listed in Table 3 are possible stored seed pathogens capable of causing deterioration and reduction of quality of the stored seed. Species of Aspergillus and Penicillium would be suspect in the light of Christensen's work with wheat (3).

GENERAL DISCUSSION

In the United States there is almost a complete lack of published information on the incidence of seed-borne pathogens associated with both agricultural and horticultural crops. It is the writer's hope that this study, which has been confined to eight species of forage legumes, may stimulate investigators to examine critically seed produced in other areas. Precise data on the infestation of various seeds with pathogens would enable pathologists to judge the general effectiveness of seeds as natural sources of disease, and to choose areas favorable for production of "disease-free" seed. In addition, such information would indicate need for seed treatment.

Investigation of the microfloras of seed offers a wonderful training ground for students of mycology and should not be overlooked by investigators for students under their guidance.

CONCLUSIONS

1) A considerable number of phytopathogens were associated with forage legume seed produced in Oregon. Fortunately, some occurred so infrequently and at such low levels that it is unlikely that they appreciably influence the quality of Oregon seed. However, a number of fungal pathogens were repeatedly detected in seed lots; these were Mycosphaerella pinodes and

Botrytis cinerea with field pea seed; Phoma herbarum var. medicaginis, Fusarium roseum and Stemphylium botryosum with alfalfa seed; B. cinerea, F. roseum, Phoma trifolii and S. botryosum with alsike clover seed; B. cinerea, F. roseum, P. trifolii, Sclerotinia sclerotiorum and S. botryosum with crimson clover seed; and Ascochyta pisi with hairy vetch seed. These latter pathogens are considered important enough to merit consideration in any program designed to produce and market high quality "disease-free" seed. 2) The levels of infestation of seed lots by pathogens were generally quite low. 3) The saprophytes associated with Oregon's forage legume seed were mainly innocuous and in general probably have little direct effect on the quality of the seed. Certain saprophytic species, however, may indirectly affect seed quality by attacking seedlings during germination tests and thus influencing the accuracy of these tests. 4) This investigation has shown that even in a region such as Oregon, theoretically ideally suited for the production of "disease-free" seed because of low summer precipitation and low relative humidities, transmission of pathogens by seeds does occur.

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OREGON AGRICULTURAL EXPERIMENT STATION, CORVALLIS

FIELD RESISTANCE OF 29 ADDITIONAL STRAWBERRY VARIETIES AND SELECTIONS TO VERTICILLIUM, 1959[1]

E. H. Varney, J. N. Moore, and D. H. Scott[2]

For reasons previously noted[3], the use of resistant varieties appears to be the best measure to follow in New Jersey for control of strawberry wilt caused by Verticillium albo-atrum Reinke & Berth. This paper records the results of the field testing in 1959 of an additional 29 varieties or selections for sources of resistance.

METHODS

Dormant strawberry plants were set April 17, 1959 in plots adjacent to the 1958 test area. Each of four randomized blocks included the varieties and selections listed in Table 1, and each plot consisted of 12 plants spaced 1 foot apart. Jerseybelle and Dixieland were included as standards.

Plants were rated for wilt on August 4 and 26, September 21, and November 4, 1959. The disease classes recognized were 0, plant apparently healthy; 1, slight symptoms, no necrosis; 2, moderate symptoms, some necrosis; 3 extensive necrosis; 4, plant nearly dead; 5, plant dead. Since 1 or 2 plants failed to grow in many of the plots, the last 10 plants in each plot were arbitrarily selected for rating. A disease index for each plot was obtained by totaling the individual ratings of the 10 plants. Mean plot values were converted after analysis to a 0 to 100 scale, in which 0 indicates all plants apparently healthy and 100 all plants dead.

US-4355, US-4356, Md-US-2611, and Md-683 were included in the test, but too few plants survived the spring setting to permit use of the data obtained in the statistical analysis. Data for these selections are, however, included in Table 1 as indicated.

Table 1. Field evaluation of various strawberry varieties and selections for susceptibility to Verticillium in New Jersey, November 4, 1959.

Variety or selection	% dead or severely wilted	Disease index[a]	Parentage
Cavalier	0.0	1.0	Valentine x Sparkle
Siletz	0.0	2.0	2 Oregon selections
NC-2492	2.5	4.0	Albritton x Md-US-2101
Md-US-2700	5.0	5.5	Pocahontas x Stelemaster
Robinson	5.0	6.0	Howard 17 (Premier) x Washington
Md-683[b]	7.4	6.7[b]	Scotland BK 46 x Fairfax
Md-US-2640	5.0	8.5	B 83.5 x Stelemaster
Grenadier	5.0	10.0	Valentine x Fairfax
NC-2475	12.5	13.5	Albritton x Md-US-2101
NC-2480	17.5	16.5	Albritton x Md-US-2101
Headliner	15.4	18.5	L 7-27 x L 7-42
Md-US-2611[b]	15.6	21.9[b]	US-4152 x Stelemaster
NC-2488	25.0	22.5	Albritton x Md-US-2101
US-3563	20.0	23.5	F. virginiana Sheld. x Midland
Md-US-2590	28.9	27.0	US-4152 x Stelemaster
Md-US-2101	25.0	31.0	Midland x Md-683
Md-US-2632	32.5	32.0	Stelemaster x Redglow

[1]Paper of the Journal Series, New Jersey Agricultural Experiment Station, Rutgers University, the State University of New Jersey, New Brunswick.

[2]Associate Professor, Research Associate, New Jersey Agricultural Experiment Station; and Principal Horticulturist, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, respectively.

[3]Varney, E. H., J. N. Moore, and D. H. Scott. 1959. Field resistance of various strawberry varieties and selections to Verticillium. Plant Disease Reptr. 43: 567-569.

Table 1. (continued)

Variety or selection	% dead or severely wilted	Disease index[a]	Parentage
NC-2605	35.0	34.5	NC-1487 x NC-1672
US-4356[b]	34.5	36.6[b]	Dixieland x Albritton
US-4355[b]	36.6	38.0[b]	Dixieland x Albritton
NC-2485	45.0	40.0	Albritton x Md-US-2101
NC-2341	47.5	44.0	NC-1430 x Albritton
Md-US-2664	42.5	46.5	Redglow x Md-US-2210
NC-2494	48.6	48.0	Albritton x Md-US-2101
Dixieland	59.0	55.0	Tennessee Shipper x Midland
NC-2484	60.0	60.0	Albritton x Md-US-2101
Jerseybelle	67.6	61.0	NJ-953 x NJ-925
Md-US-2699	63.2	63.0	Pocahontas x Stelemaster
NJ-157	62.5	64.5	Utah Shipper x Jerseybelle
NC-2478	75.0	73.5	Albritton x Md-US-2101
NJ-257	85.0	83.0	Jerseybelle x Albritton
L.S.D. 19:1		16.8	
L.S.D. 99:1		22.3	

[a] Based on scale in which 0 indicates all plants apparently healthy and 100 all plants dead.

[b] Data for Md-683, Md-US-2611, US-4356, and US-4355 based on 27, 32, 29, and 30 plants, respectively, were not included in the statistical analysis.

RESULTS AND DISCUSSION

Although wilt in the test area was not so severe as in 1958, the results (Table 1) clearly indicate the varieties or selections that will be of value in the breeding program. Cavalier and Siletz are of particular interest, for none of the plants developed severe wilt symptoms. Selections NC-2492, Md-US-2700, and Md-US-2640 and varieties Robinson and Grenadier also were highly resistant. The standards, Jerseybelle and Dixieland, were again highly susceptible. Resistance and susceptibility of varieties and selections graded into each other, indicating again a quantitative type of inheritance[3].

Eight selections from a cross of intermediately resistant parents (Albritton x Md-US-2101) varied greatly in their reaction to Verticillium. NC-2492 was highly resistant; NC-2475, NC-2480 and NC-2488 were moderately resistant; and NC-2485, NC-2494, NC2484, and NC-2478 were moderately to highly susceptible. The fact that these eight selections ranged in reaction from highly resistant to highly susceptible suggests that intermediate parents with superior horticultural characteristics should not be ignored in the breeding program for wilt resistance.

NEW JERSEY AGRICULTURAL EXPERIMENT STATION, NEW BRUNSWICK

DOWNY MILDEW ON WATERMELONS IN ARIZONA, A FIRST REPORT

Chester R. Leathers

During the late summer of 1958, a report of a severe outbreak of anthracnose on Charleston Gray watermelons at the farm of Mr. O. C. McDaniel of the Double-Adobe District, 10 miles north of Douglas, Arizona was received by Dr. Thomas W. Barrett, Professor of Agronomy at Arizona State University. With Dr. Barrett's assistance, the field was surveyed on September 27, 1958, at which time many of the leaves were found to be badly wilted while others were flat and crisp or markedly dried out in the terminal portion of the lobes. Nearly all leaves exhibited abundant, angular, brownish to blackish spots approximately 1 cm in diameter over the upper surface. No lesions were found on either vines or fruits although the fruits were badly sunburned. Microscopic examination of the diseased leaves revealed abundant conidia and conidiophores of Pseudoperonospora cubensis (Berk. & Curt.) Rostow. in the region of the discolored lesions. No evidence of the anthracnose organism was found. Infected material was sent to Dr. G. H. Boewe and Dr. W. C. Snyder for verification of the pathogen's identity.

A thorough search of the same field was made throughout the summer and fall of 1959 but no evidence of downy mildew was obtained.

Abundant records and collections of downy mildew on cucumbers and cantaloupes have been made from all areas of the State where such crops are grown but no reports have heretofore been made of its occurrence on watermelon in Arizona.

Pseudoperonospora cubensis was reported as widespread on watermelons during the spring of 1958 in the Yaqui Valley of Mexico[1] and infections could presumably have occurred by airborne inoculum from this region.

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[1]Yerkes, William D., Jr., J. S. Niederhauser, N. E. Borlaug, Eugenio Martinez S., and Jorge Galindo A. 1959. Some plant diseases observed in Mexico in 1958. Plant Disease Reptr. 43: 500-503.

STEWART'S DISEASE: EXPECTED DEVELOPMENT IN ILLINOIS IN 1960

G. H. Boewe

Stewart's disease (Bacterium stewartii E. F. Sm.), or bacterial wilt, of corn is expected to be more destructive and to occur much farther north in Illinois in the summer of 1960 than it did in the summers of 1958 and 1959. This prediction is based on the close correlation that apparently exists between the winter temperatures and the amount of disease that develops during the following summer.

Weatherwise the winter of 1959-1960 in Illinois was unusual. The cold weather which began in October continued through November. Mean temperatures of November were from 5 to 6 degrees and 6 to 8 degrees below normal in the south half and north half of the State respectively. On the morning of the 17th temperatures dropped to slightly below zero to 5 degrees above in the north half and from 5 to 10 degrees above in the south half of the State. These were the lowest temperatures ever recorded in the State so early in the season. Following the very cold November, December was unusually warm; mean temperatures ranged from 6 to 8 degrees above normal in the north half and 3 to 6 degrees above normal in the south half of the State. Mean temperatures of January were about normal; those of February averaged 4.2 degrees below normal for the State. The coldest temperatures of the winter occurred in late February, but the entire State had a good snow cover.

Based on the winter indexes (sum of the mean temperature of December, January and February) for 91 weather reporting stations in Illinois, the early wilt phase of the Stewart's disease on susceptible varieties of sweet corn is expected to be destructive in the south 2/5 of the State. North of this area, in a band about 100 miles wide, wilt is expected to be moderately severe. It probably will be light or absent in the rest of the State. In the northern three tiers of counties, except in those counties bordering Lake Michigan, no wilt, or only a trace of wilt is anticipated.

The late season leaf blight phase of Stewart's disease is expected to be moderate to severe in the south half of the State, light in the north-central part, and only a trace in the remainder of the State.

ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA, ILLINOIS

SOOTY-BARK CANKER OF ASPEN IN NEW MEXICO

Stuart R. Andrews and Wallace E. Eslyn[1]

In September 1959 sooty-bark canker of aspen (Populus tremuloides) was observed for the first time in New Mexico. Several cankered trees were present in a declining stand on Chuska Mountain on the Navajo Indian Reservation in the northwestern corner of the State. A month later the canker was found in other aspen stands about 180 miles due east on Elk Mountain in the Upper Pecos District of the Santa Fe National Forest. Occurrence of the canker in such widely separated localities suggests that it may be common in New Mexico.

Davidson and Cash[2] first reported the sooty-bark canker in 1955 and stated that it was rather uniformly distributed throughout older or mature aspen stands in Colorado. These authors found a discomycete consistently associated with the canker, which they renamed Cenangium singulare (Rehm.) comb. n. The fungus was also associated with all old cankers examined in New Mexico.

Discovery of the sooty-bark canker on Chuska and Elk Mountains coincided with reports of abnormally heavy mortality in mature and overmature aspen stands on the Santa Fe National Forest, but enough field observations have not been made to determine whether this particular canker may be a factor in the dying. However, it is considered highly suspect because inoculation tests in Colorado[3] have demonstrated the pathogenicity of C. singulare, especially its rapid girdling action.

ROCKY MOUNTAIN FOREST AND RANGE EXPERIMENT STATION, FOREST SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

[1]Plant Pathologists, Rocky Mountain Forest and Range Experiment Station, Forest Service, United States Department of Agriculture. General headquarters at Colorado State University in Fort Collins, Colorado.

[2]Davidson, Ross W., and Edith K. Cash. 1956. A Cenangium associated with sooty-bark canker of aspen. Phytopathology 46: 34-36.

[3]Hinds, T. E., and F. G. Hawksworth. 1958. Inoculation tests with Cenangium singulare. Progress Rept. U. S. Forest Service, Rocky Mountain Forest and Range Experiment Station. 3 pp. (Typewritten report).

REFERENCE TO "MELILOTUS ITALICA, A NEW HOST FOR UROMYCES STRIATUS"

I. L. Conners

In the March 15 issue of the Plant Disease Reporter, E. E. Leppik reports the occurrence of Uromyces striatus on Melilotus italica, but in his discussion he states that "no pycnia or aecia have ever been found in America." This statement is in error for aecia of this rust, sub U. medicaginis, were collected on Euphorbia cyperissias and uredinia on Medicago lupulina and alfalfa at Braeside, Renfrew Co., Ontario, in 1947 (Can. Plant Disease Survey Ann. Rept. 27: 24-25. 1948). Greenhouse inoculations confirmed the observations in the field. Also the records of the Mycological Herbarium, Plant Research Institute, Ottawa reveal that duplicate specimens of the rust on E. cyperissias and M. sativa from Braeside and Hornings Mills, Dufferin Co., Ont., have been deposited in the Herbarium at Beltsville. Admittedly U. striatus occurs less frequently than the autoecious rust, Melampsora euphorbiae, in Ontario, but surely there are suitable locations in the United States where U. striatus may be found on Euphorbia.

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